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=> fil reg; d ide
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DICTIONARY FILE UPDATES:  16 JAN 2007  HIGHEST RN 917560-96-4

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L11  ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2007 ACS on STN
RN   99085-47-9  REGISTRY
ED   Entered STN:  16 Nov 1985
CN   Complement decay-accelerating factor (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   CD55 antigen
CN   DAF
CN   Decay-accelerating factor
CN   Decay-accelerating factor glycoproteins
CN   Glycoproteins (specific proteins and subclasses), DAF
MF   Unspecified
CI   MAN
SR   CA
LC   STN Files:   ADISNEWS, AGRICOLA, BIOSIS, BIOTECHNO, CA, CAPLUS, CIN,
       EMBASE, PHAR, PROMT, TOXCENTER, USPAT2, USPATFULL

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

            1079 REFERENCES IN FILE CA (1907 TO DATE)
              29 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
            1086 REFERENCES IN FILE CAPLUS (1907 TO DATE)
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INVENTOR SEARCH

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=> => fil jic pascal biotechno biosis esbio biotechds lifesci confsci dissabs
bioeng scisearch
FILE 'JICST-EPLUS' ENTERED AT 16:54:35 ON 17 JAN 2007
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FILE 'SCISEARCH' ENTERED AT 16:54:35 ON 17 JAN 2007
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=> d que l76

L67          306 SEA VOLLMERS H?/AU
L68        10926 SEA MUELLER HERMELINK H?/AU OR MUELLER H?/AU OR HERMELINK
                 H?/AU
L69         7655 SEA CD55 OR CD 55 OR DECAY ACCELERATING FACTOR
L71           51 SEA 23132?
L76           14 SEA (L67 AND L68) AND (L69 OR L71)


=> fil medl; d que l25

FILE 'MEDLINE' ENTERED AT 16:54:37 ON 17 JAN 2007

 FILE LAST UPDATED: 16 Jan 2007 (20070116/UP).  FILE COVERS 1950 TO DATE.

  All regular MEDLINE updates from November 15 to December 16 have been
  added to MEDLINE, along with 2007 Medical Subject Headings (MeSH(R))
  and 2007 tree numbers.
```

The annual reload will be available in early 2007

This file contains CAS Registry Numbers for easy and accurate substance identification.

L23 674 SEA FILE=MEDLINE ABB=ON MUELLER HERMELINK H?/AU OR MUELLER H?/AU OR HERMELINK H?/AU
L24 50 SEA FILE=MEDLINE ABB=ON VOLLMERS H?/AU
L25 1 SEA FILE=MEDLINE ABB=ON L23 AND L24

=> fil embase; d que l46

FILE 'EMBASE' ENTERED AT 16:54:38 ON 17 JAN 2007

FILE COVERS 1974 TO 17 Jan 2007 (20070117/ED)

EMBASE is now updated daily. SDI frequency remains weekly (default) and biweekly.

This file contains CAS Registry Numbers for easy and accurate substance identification.

L37 53 SEA FILE=EMBASE ABB=ON VOLLMERS H?/AU
L38 896 SEA FILE=EMBASE ABB=ON MUELLER HERMELINK H?/AU OR MUELLER H?/AU OR HERMELINK H?/AU
L39 1317 SEA FILE=EMBASE ABB=ON DECAY ACCELERATING FACTOR/CT
L46 8 SEA FILE=EMBASE ABB=ON (L37 AND L38) OR ((L37 OR L38) AND L39)

=> fil wpix; d que l53

FILE 'WPIX' ENTERED AT 16:54:39 ON 17 JAN 2007

FILE LAST UPDATED: 15 JAN 2007 <20070115/UP>
MOST RECENT THOMSON SCIENTIFIC UPDATE: 200704 <200704/DW>
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

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>>> IPC Reform reclassification data for the backfile is being loaded into the database during the first half of January 2007. There will not be any update date (UP) written for the reclassified documents, but they can be identified by 20060101/UPIC. <<<

FOR A COPY OF THE DERWENT WORLD PATENTS INDEX STN USER GUIDE, PLEASE VISIT:
http://www.stn-international.de/training_center/patents/stn_guide.pdf

FOR DETAILS OF THE PATENTS COVERED IN CURRENT UPDATES, SEE
http://scientific.thomson.com/support/patents/coverage/latestupdates/

PLEASE BE AWARE OF THE NEW IPC REFORM IN 2006, SEE
http://www.stn-international.de/stndatabases/details/ipc_reform.html and
http://scientific.thomson.com/media/scpdf/ipcrdwpi.pdf

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>>> FOR DETAILS ON THE NEW AND ENHANCED DERWENT WORLD PATENTS INDEX
    PLEASE SEE
http://www.stn-international.de/stndatabases/details/dwpi_r.html <<<

>>> New and revised Manual Codes went live in Derwent World Patents Index
    To view the lists of new, revised and retired codes for both CPI and
    EPI,  please go to:
    http://scientific.thomson.com/dwpi-manualcoderevision <<<
'BI ABEX' IS DEFAULT SEARCH FIELD FOR 'WPIX' FILE


L50          14 SEA FILE=WPIX ABB=ON  VOLLMERS H?/AU
L51        2606 SEA FILE=WPIX ABB=ON  MUELLER HERMELINK H?/AU OR MUELLER H?/AU
                OR HERMELINK H?/AU
L52         119 SEA FILE=WPIX ABB=ON  CD55/BI,ABEX OR CD 55/BI,ABEX OR DECAY
                ACCELERATING FACTOR/BI,ABEX
L53           1 SEA FILE=WPIX ABB=ON  (L50 AND L51) OR ((L50 OR L51) AND L52)


=> fil capl; d que l5; d que l9; s l5 or l9

FILE 'CAPLUS' ENTERED AT 16:54:40 ON 17 JAN 2007
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FILE COVERS 1907 - 17 Jan 2007  VOL 146 ISS 4
FILE LAST UPDATED: 16 Jan 2007  (20070116/ED)

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They are available for your review at:

http://www.cas.org/infopolicy.html
'OBI' IS DEFAULT SEARCH FIELD FOR 'CAPLUS' FILE


L1           48 SEA FILE=CAPLUS ABB=ON  VOLLMERS H?/AU
L2         5844 SEA FILE=CAPLUS ABB=ON  MUELLER HERMELINK H?/AU OR MUELLER
                H?/AU OR HERMELINK H?/AU
L3           25 SEA FILE=CAPLUS ABB=ON  L1 AND L2
L4          362 SEA FILE=CAPLUS ABB=ON  CD55/OBI OR CD 55/OBI
L5            2 SEA FILE=CAPLUS ABB=ON  L3 AND L4



L1           48 SEA FILE=CAPLUS ABB=ON  VOLLMERS H?/AU
L2         5844 SEA FILE=CAPLUS ABB=ON  MUELLER HERMELINK H?/AU OR MUELLER
```

H?/AU OR HERMELINK H?/AU

L7 4 SEA FILE=CAPLUS ABB=ON 23132/OBI
L9 4 SEA FILE=CAPLUS ABB=ON (L1 OR L2) AND L7

L80 6 L5 OR L9

=> dup rem l25,l80,l53,l76,l46
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PROCESSING COMPLETED FOR L25
PROCESSING COMPLETED FOR L80
PROCESSING COMPLETED FOR L53
PROCESSING COMPLETED FOR L76
PROCESSING COMPLETED FOR L46
L81 22 DUP REM L25 L80 L53 L76 L46 (8 DUPLICATES REMOVED)
ANSWER '1' FROM FILE MEDLINE
ANSWERS '2-7' FROM FILE CAPLUS
ANSWER '8' FROM FILE WPIX
ANSWERS '9-11' FROM FILE PASCAL
ANSWERS '12-15' FROM FILE BIOSIS
ANSWER '16' FROM FILE BIOTECHDS
ANSWERS '17-22' FROM FILE EMBASE

=> d ibib ed abs 1-22

L81 ANSWER 1 OF 22 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 96084039 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 7492750
TITLE: Efficient immortalization of rheumatoid synovial tissue B-lymphocytes. A comparison between the techniques of electric field-induced and PEG fusion.
AUTHOR: Krenn V; von Landenberg P; Wozniak E; Kissler C; **Hermelink H K**; Zimmermann U; **Vollmers H P**
CORPORATE SOURCE: Institut fur Pathologie, Universitat Wurzburg, Germany.
SOURCE: Human antibodies and hybridomas, (1995) Vol. 6, No. 2, pp.

47-51.
Journal code: 9014461. ISSN: 0956-960X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199601
ENTRY DATE: Entered STN: 17 Feb 1996
Last Updated on STN: 17 Feb 1996
Entered Medline: 11 Jan 1996
ED Entered STN: 17 Feb 1996
Last Updated on STN: 17 Feb 1996
Entered Medline: 11 Jan 1996
AB In this study, B-cells isolated from rheumatoid synovial tissue were immortalized, without prior in vitro stimulation, by means of electric-field induced fusion and conventional PEG fusion in order to compare the efficiency of these methods. Two myeloma cell lines were used as fusion partners, the murine myeloma Ag8 and the murine-human heteromyeloma HAB-1. The results of seven fusion experiments performed simultaneously with identical cell populations showed that fusion frequencies obtained by electrofusion were 4 to 35 times higher than by the PEG fusion technique. The morphological and immunohistochemical evaluation of synovial tissues used for fusion showed that only tissues exhibiting a follicular distribution of B-cells with a high percentage of CD 22-positive lymphocytes gave rise to high fusion yields and produced B-cell clones, whereas synovial tissues with the same percentage of plasma cells but lower percentages of CD 22 lymphocytes yielded very low fusion rates. In conclusion, electrofusion is more efficient for immortalizing small amounts of synovial tissue B-lymphocytes than PEG fusion, since high fusion frequencies could be obtained by this technique without the need for prior in vitro stimulation. Synovial tissue exhibiting a follicular distribution of B-lymphocytes with high percentages of CD 22-positive lymphocytes gave rise to high hybridoma yields and therefore an ideal source of human rheumatoid B-cell clones.

L81 ANSWER 2 OF 22 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2005:1289419 CAPLUS Full-text
DOCUMENT NUMBER: 144:35307
TITLE: CFR-1 isoform-binding polypeptides or antibodies and conjugates for diagnosis and treatment of cancer
INVENTOR(S): **Vollmers, Heinz Peter**; **Mueller-Hermelink, Hans Konrad**; Hensel, Frank
PATENT ASSIGNEE(S): Debiovision Inc., Can.
SOURCE: PCT Int. Appl., 131 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005116076	A2	20051208	WO 2005-IB2480	20050126
WO 2005116076	A3	20060406		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2005032134	A1	20050210	US 2004-764730	20040126
CA 2553826	A1	20051208	CA 2005-2553826	20050126
EP 1711525	A2	20061018	EP 2005-780003	20050126

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, BA, HR, IS, YU

PRIORITY APPLN. INFO.:		
US 2004-764730	A	20040126
DE 2001-10136009	A	20010724
DE 2002-10210425	A	20020309
WO 2002-DE2699	A2	20020723
WO 2005-IB2480	W	20050126

ED Entered STN: 09 Dec 2005

AB The present invention features novel polypeptides and methods of using these polypeptides in the diagnosis, detection, monitoring, and treatment of neoplasms in mammal, e.g., a human. The polypeptides are neoplasm-specific polypeptides or antibodies specific to novel isoform of CFR-1 that is expressed on neoplastic cells as well as cells of pre-cancerous lesions but not normal cells.

L81 ANSWER 3 OF 22 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 2

ACCESSION NUMBER: 2005:1103416 CAPLUS Full-text
DOCUMENT NUMBER: 143:385158
TITLE: Identification and purification of human tumor-specific polypeptides or antibodies from healthy donors for cancer diagnosis and therapy
INVENTOR(S): **Vollmers, Heinz Peter;**
Mueller-Hermelink, Hans Konrad
PATENT ASSIGNEE(S): Oncomab G.m.b.H., Germany
SOURCE: PCT Int. Appl., 79 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005094159	A2	20051013	WO 2004-IB4453	20041112

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2004317819	A1	20051013	AU 2004-317819	20041112
CA 2545512	A1	20051013	CA 2004-2545512	20041112

PRIORITY APPLN. INFO.:		
US 2003-519550P	P	20031112
WO 2004-IB4453	W	20041112

ED Entered STN: 14 Oct 2005

AB The present invention features methods of identifying, from healthy donors, polypeptides, such as antibodies, that are specific for neoplasm, polypeptides identified using such methods, and their use in the treatment and diagnosis of neoplasms.

L81 ANSWER 4 OF 22 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 7
ACCESSION NUMBER: 1994:128916 CAPLUS Full-text
DOCUMENT NUMBER: 120:128916
TITLE: Characterization of four new gastric cancer cell lines
AUTHOR(S): **Vollmers, H. Peter**; Stulle, Konrad; Daemmrich, Jobst; Pfaff, Martin; Papadopoulos, T.; Betz, Christoph; Saal, Katharina; **Mueller-Hermelink, Hans Konrad**
CORPORATE SOURCE: Inst. Pathol., Wuerzburg, W-8700, Germany
SOURCE: Virchows Archiv B: Cell Pathology Including Molecular Pathology (1993), 63(6), 335-343
CODEN: VAAZA2; ISSN: 0340-6075
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 19 Mar 1994
AB Four well differentiated gastric adenocarcinoma cell lines from German patients have been established from primary tumors (St 23132, St 3051) and lymph node metastases (St 2474, St 2957). The tumor cells were isolated by enzymic or mech. treatment. All four lines grew as solid tumors in nude mice and formed colonies in soft agar. The doubling time of the cells in culture was 25-32 h. Further characteristics of the lines were a considerable chromosomal aneuploidy, (the chromosomal nos. varying from 30-109 with many numerical and structural abnormalities), a stable keratin expression (Ck 8, 18, 19), the expression and secretion of CEA and CA-19-9 and the overexpression of c-myc. The four stomach cancer cell lines described here are not only a useful addition to the small number of existing lines, but also represent ideal tools for studying tumorigenicity of human stomach cancers in vitro and in vivo.

L81 ANSWER 5 OF 22 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:451503 CAPLUS Full-text
DOCUMENT NUMBER: 143:6288
TITLE: Anti-idiotype antibodies for human monoclonal antibody SC-1
INVENTOR(S): **Vollmers, Heinz Peter**; **Mueller-Hermelink, Hans Konrad**
PATENT ASSIGNEE(S): H3 Pharma Inc., Can.; Debiovision Inc.
SOURCE: PCT Int. Appl., 27 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005047456	A2	20050526	WO 2004-IB4407	20041115
WO 2005047456	A3	20060323		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,

TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

DE 10352977	A1	20050609	DE 2003-10352977	20031113
AU 2004288870	A1	20050526	AU 2004-288870	20041115
CA 2546323	A1	20050526	CA 2004-2546323	20041115
EP 1694708	A2	20060830	EP 2004-806561	20041115

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR, IS, YU

PRIORITY APPLN. INFO.:
DE 2003-10352977 A 20031113
WO 2004-IB4407 W 20041115

ED Entered STN: 27 May 2005

AB The authors disclose the preparation and characterization of anti-idiotype antibodies for the human monoclonal antibody SC-1.

L81 ANSWER 6 OF 22 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:451421 CAPLUS Full-text
DOCUMENT NUMBER: 143:6286
TITLE: Adenocarcinoma-specific antibody SAM-6, fragments and conjugates for cancer diagnosis and therapy
INVENTOR(S): **Vollmers, Heinz; Mueller-Hermelink, Hans-Konrad**
PATENT ASSIGNEE(S): Oncomab G.m.b.H., Germany
SOURCE: PCT Int. Appl., 104 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005047332	A1	20050526	WO 2004-EP12970	20041112

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1531162	A1	20050518	EP 2003-26161	20031114

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

AU 2004289821	A1	20050526	AU 2004-289821	20041112
CA 2545454	A1	20050526	CA 2004-2545454	20041112
EP 1709083	A1	20061011	EP 2004-797921	20041112

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, IS

PRIORITY APPLN. INFO.:
EP 2003-26161 A 20031114
DE 2002-10229906 A 20020704
WO 2004-EP12970 W 20041112

ED Entered STN: 27 May 2005
AB The present invention features a polypeptide, such as an antibody produced by the hybridoma SAM-6 and its use in the treatment and diagnosis of neoplasms.
REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L81 ANSWER 7 OF 22 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2000:441814 CAPLUS Full-text
DOCUMENT NUMBER: 133:53690
TITLE: Substance for producing highly effective antitumor medicaments
INVENTOR(S): **Vollmers, Heinz Peter;**
Mueller-Hermelink, Hans Konrad
PATENT ASSIGNEE(S): Germany
SOURCE: PCT Int. Appl., 54 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2000037489	A2	20000629	WO 1999-EP10329		19991222
WO 2000037489	A3	20001109			
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW					
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG					
DE 19909771	A1	20000629	DE 1999-19909771		19990305
CA 2356189	A1	20000629	CA 1999-2356189		19991222
EP 1141019	A2	20011010	EP 1999-969227		19991222
EP 1141019	B1	20040421			
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO					
JP 2002534357	T	20021015	JP 2000-589558		19991222
AU 772579	B2	20040429	AU 2000-27955		19991222
AT 264873	T	20040515	AT 1999-969227		19991222
PT 1141019	T	20040930	PT 1999-969227		19991222
ES 2219108	T3	20041116	ES 1999-969227		19991222
AU 2004203479	A1	20040826	AU 2004-203479		20040729
PRIORITY APPLN. INFO.:			DE 1998-19859248	A	19981222
			DE 1999-19909771	A	19990305
			WO 1999-EP10329	W	19991222

ED Entered STN: 30 Jun 2000
AB A gastric carcinoma-specific isoform of glycoprotein CD55/DAF containing a tumor-specific carbohydrate structure is obtained from membrane prepns. from human adenocarcinoma cell line 23132 and used to screen candidate tumor-binding and apoptosis-inducing substances for their ability to bind to the CD55/DAF isoform, to determine their potential usefulness in tumor diagnosis and therapy. The test substances may be peptides, peptidomimetics, antibodies, antibody fragments, or antibody derivs. (except for monoclonal antibody SC-1 directed to CD55).

L81 ANSWER 8 OF 22 WPIX COPYRIGHT 2007 THE THOMSON CORP on STN

ACCESSION NUMBER: 2000-477068 [42] WPIX
DOC. NO. CPI: C2000-143524 [42]
TITLE: New glycoprotein with tumor-specific glycosylation, useful in screening for agents for treating or diagnosing tumors, contains the **CD55** primary amino acid sequence
DERWENT CLASS: B04; D16
INVENTOR: MUELLER-HERMELINK H K; MULLER-HERMELINK H K; VOELLMERS H P; **VOLLMERS H P**
PATENT ASSIGNEE: (MUEL-I) MUELLER-HERMELINK H K; (MULL-I) MULLER-HERMELINK H; (MULL-I) MULLER-HERMELINK H K; (VOLL-I) VOLLMERS H; (VOLL-I) VOLLMERS H P
COUNTRY COUNT: 87

PATENT INFO ABBR.:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
DE 19909771	A1	20000629	(200042)*	DE	22	[13]
WO 2000037489	A2	20000629	(200042)	DE		
AU 2000027955	A	20000712	(200048)	EN		
EP 1141019	A2	20011010	(200167)	DE		
JP 2002534357	W	20021015	(200282)	JA	49	
EP 1141019	B1	20040421	(200428)	DE		
DE 59909264	G	20040527	(200436)	DE		
AU 772579	B2	20040429	(200457)	EN		
AU 2004203479	A1	20040826	(200476)#	EN		
ES 2219108	T3	20041116	(200477)	ES		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
DE 19909771	A1	DE 1999-19909771	19990305
DE 59909264	G	DE 1999-59909264	19991222
EP 1141019	A2	EP 1999-969227	19991222
EP 1141019	B1	EP 1999-969227	19991222
DE 59909264	G	EP 1999-969227	19991222
ES 2219108	T3	EP 1999-969227	19991222
WO 2000037489	A2	WO 1999-EP10329	19991222
EP 1141019	A2	WO 1999-EP10329	19991222
JP 2002534357	W	WO 1999-EP10329	19991222
EP 1141019	B1	WO 1999-EP10329	19991222
DE 59909264	G	WO 1999-EP10329	19991222
AU 2000027955	A	AU 2000-27955	19991222
AU 772579	B2	AU 2000-27955	19991222
JP 2002534357	W	JP 2000-589558	19991222
AU 2004203479	A1	AU 2004-203479	20040729

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 772579	B2	Previous Publ	AU 2000027955 A
AU 2004203479	A1	Div ex	AU 772579 B
DE 59909264	G	Based on	EP 1141019 A
ES 2219108	T3	Based on	EP 1141019 A
AU 2000027955	A	Based on	WO 2000037489 A
EP 1141019	A2	Based on	WO 2000037489 A
JP 2002534357	W	Based on	WO 2000037489 A

EP 1141019 B1 Based on WO 2000037489 A
DE 59909264 G Based on WO 2000037489 A
AU 772579 B2 Based on WO 2000037489 A

PRIORITY APPLN. INFO: DE 1998-19859248 19981222
DE 1999-19909771 19990305
AU 2004-203479 20040729

ED 20050705
AN 2000-477068 [42] WPIX
AB DE 19909771 A1 UPAB: 20050705
NOVELTY - A glycoprotein (I) which comprises at least a segment of the primary amino acid sequence of **CD55** and has a tumor-specific pattern of glycosylation, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following: (1) a method for the recovery of (I); and (2) substances (II) that bind specifically to (I) and initiate a phosphorylation cascade.
ACTIVITY - Cytostatic.
MECHANISM OF ACTION - Agents that bind specifically to (I) induce apoptosis and/or initiate a phosphorylation cascade.
USE - (I) are used to screen for its specific binding agents (II), particularly those that recognize its specific sugar structure. (II) are used for the induction of apoptosis, for the treatment or diagnosis (including imaging) of tumors and to initiate a **CD55**-mediated phosphorylation cascade (all claimed).

Member(0002)
ABEQ WO 2000037489 A2 UPAB 20050705
NOVELTY - A glycoprotein (I) which comprises at least a segment of the primary amino acid sequence of **CD55** and has a tumor-specific pattern of glycosylation, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) a method for the recovery of (I); and
(2) substances (II) that bind specifically to (I) and initiate a phosphorylation cascade.
ACTIVITY - Cytostatic.
MECHANISM OF ACTION - Agents that bind specifically to (I) induce apoptosis and/or initiate a phosphorylation cascade.
USE - (I) are used to screen for its specific binding agents (II), particularly those that recognize its specific sugar structure. (II) are used for the induction of apoptosis, for the treatment or diagnosis (including imaging) of tumors and to initiate a **CD55**-mediated phosphorylation cascade (all claimed).

L81 ANSWER 9 OF 22 PASCAL COPYRIGHT 2007 INIST-CNRS. ALL RIGHTS RESERVED.
on STN DUPLICATE 3
ACCESSION NUMBER: 2002-0248437 PASCAL Full-text
COPYRIGHT NOTICE: Copyright .COPYRGT. 2002 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Regulation of the new coexpressed **CD55** (**decay-accelerating factor**) receptor on stomach carcinoma cells involved in antibody SC-1-induced apoptosis
AUTHOR: HENSEL Frank; HERMANN Ralph; BRAENDLEIN Stephanie; KRENN Veit; SCHMAUSSER Bernd; GEIS Steffen; **MUELLER-HERMELINK Hans Konrad**; VOLLMERS **H. Peter**
CORPORATE SOURCE: Institute for Pathology, University of Wuerzburg, Wuerzburg, Germany, Federal Republic of

SOURCE: Laboratory investigation; (2001), 81(11), 1553-1563, refs. 1 p. 1/2
ISSN: 0023-6837 CODEN: LAINAW
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-8078, 354000100017670080
UP 20020604
AN 2002-0248437 PASCAL Full-text
CP Copyright .COPYRGT. 2002 INIST-CNRS. All rights reserved.
AB The human monoclonal antibody SC-1 was isolated from a patient with a diffuse-type adenocarcinoma of the stomach using somatic cell hybridization. The immunoglobulin (Ig)M antibody reacts specifically with diffuse- (70%) and intestinal-type (25%) gastric adenocarcinoma and induces apoptosis in vitro and in vivo. When used in clinical trials with stomach carcinoma patients, significant apoptotic and regressive effects in primary tumors have been observed with the antibody SC-1, The SC-1 receptor is a new 82 kd membrane-bound isoform of glycosylphosphatidylinositol (GPI)-linked **CD55** (**decay - accelerating factor**, DAF). **CD55** is known to protect cells from lysis through autologous complement and is coexpressed with the ubiquitously distributed 70 kd isoform. The SC-1-specific **CD55** isoform is up-regulated shortly after antibody binding, followed by an internalization of the antibody/receptor-complex, whereas the membranous expression of wild-type **CD55** remains unchanged. The apoptotic process is marked by cleavage of cytokeratin 18, indicating the involvement of caspase-6 in the apoptotic process. In contrast to other apoptotic pathways, a cleavage of poly(ADP-ribose)polymerase (PARP) is not observed. The expression of the cell-cycle regulator c-myc becomes up-regulated, whereas expression of topoisomerase IIα is down-regulated. Induction of apoptosis leads to an increase in the internal Ca.sup.2.sup.+ concentration, which is not necessary for the apoptotic process but for the transport of newly synthesized SC-1-specific **CD55** isoform to the membrane.

L81 ANSWER 10 OF 22 PASCAL COPYRIGHT 2007 INIST-CNRS. ALL RIGHTS RESERVED.
on STN DUPLICATE 4
ACCESSION NUMBER: 2000-0003046 PASCAL Full-text
COPYRIGHT NOTICE: Copyright .COPYRGT. 2000 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Characterization of glycosylphosphatidylinositol-linked molecule **CD55/decay-accelerating factor** as the receptor for antibody SC-1-induced apoptosis
AUTHOR: HENSEL F.; HERMANN R.; SCHUBERT C.; ABE N.; SCHMIDT K.; FRANKE A.; SHEVCHENKO A.; MANN M.; **MUELLER-HERMELINK H. K.; VOLLMERS H. P.**
CORPORATE SOURCE: Institut fuer Pathologie, 97080 Wuerzburg, Germany, Federal Republic of; European Molecular Biology Laboratory, 69012 Heidelberg, Germany, Federal Republic of
SOURCE: Cancer research : (Baltimore), (1999), 59(20), 5299-5306, 45 refs.
ISSN: 0008-5472 CODEN: CNREA8
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-5088, 354000088034140410
UP 20001101

AN 2000-0003048 PASCAL Full-text
CP Copyright .COPYRGT. 2000 INIST-CNRS. All rights reserved.
AB The human monoclonal antibody SC-1 induces apoptosis of stomach carcinoma cells and is currently used in a clinical Phase II trial. The antibody binds to a target molecule that is preferentially expressed on diffuse- and intestinal-type stomach cancer cells and shows a very restricted expression on other normal and malignant tissues. In this paper, we show that the SC-I receptor is a stomach carcinoma-associated isoform of **CD55** [membrane-bound **decay-accelerating factor** (DAF)-B] with a relative molecular mass of approximately 82 kDa. The antigenic site of SC-1 is an N-linked carbohydrate residue. Cross-linking of the DAF receptor increases apoptotic activity. SC-I binding induces tyrosine phosphorylation of three proteins of approximately 60, 75, and 110 kDa, whereas a serine residue of an approximately 35-kDa protein is dephosphorylated. Expression of caspase-3 (CPP32) and caspase-8 (FLICE) is elevated, and activation of these caspases occurs. These data show that a tumor-specific variant form DAF is involved in apoptosis and can be used for adjuvant therapeutical purposes on gastric carcinoma.

L81 ANSWER 11 OF 22 PASCAL COPYRIGHT 2007 INIST-CNRS. ALL RIGHTS RESERVED. on STN DUPLICATE 5

ACCESSION NUMBER: 1998-0072316 PASCAL Full-text
COPYRIGHT NOTICE: Copyright .COPYRGT. 1998 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Tumor-specific apoptosis induced by the human monoclonal antibody SC-1 : A new therapeutical approach for stomach cancer
AUTHOR: **VOLLMERS H. P.**; HENSEL F.; HERMANN R.; DAEMMRICH J.; WOZNIAK E.; GESSNER P.; HERRMANN B.; ZIMMERMANN U.; **MUELLER-HERMELINK H. K.**
CORPORATE SOURCE: Institut fuer Pathologie, Josef-Schneider-Str. 2, 97080 Wuerzburg, Germany, Federal Republic of; Lehrstuhl fuer Biotechnologie, Am Hubland, Universitaet Wuerzburg, 97080 Wuerzburg, Germany, Federal Republic of
SOURCE: Oncology reports, (1998), 5(1), 35-40, 25 refs. ISSN: 1021-335X
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: Greece
LANGUAGE: English
AVAILABILITY: INIST-26534, 354000077284890040
UP 20001101
AN 1998-0072316 PASCAL Full-text
CP Copyright .COPYRGT. 1998 INIST-CNRS. All rights reserved.
AB Stomach cancer is one of the most frequently occuring cancers worldwide with a very poor prognosis, even after complete gastrectomy. We describe here an alternative therapeutical approach using a human monoclonal antibody (SC-1), which was isolated from a patient with diffuse-type gastric adenocarcinoma. We demonstrate that the antibody significantly reduces stomach cancer growth in vivo, by inducing tumor-specific apoptosis and that the antibody, even delivered in high doses, shows no toxic crossreactivity to other organs or tissues. The data presented here show that tumor-specific apoptosis can be induced and they give rise to the hope that human monoclonal antibodies with biological activity might present a completely new type of adjuvant cancer therapy.

L81 ANSWER 12 OF 22 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2002:22161 BIOSIS Full-text
DOCUMENT NUMBER: PREV200200022161
TITLE: Immunotherapy for stomach carcinoma with the human monoclonal antibody SC-1: New data on CD55/SC-1 receptor signalling and apoptotic mechanisms.
AUTHOR(S): **Vollmers, H. Peter** [Reprint author]; Hensel, Frank; Hermann, Ralph; Schmausser, Bernd; Braendlein, Stephanie; **Mueller-Hermelink, Hans-Konrad**
CORPORATE SOURCE: Pathology, University of Wuerzburg, Wuerzburg, Germany
SOURCE: Proceedings of the American Association for Cancer Research Annual Meeting, (March, 2001) Vol. 42, pp. 845-846. print.
Meeting Info.: 92nd Annual Meeting of the American Association for Cancer Research. New Orleans, LA, USA. March 24-28, 2001.
ISSN: 0197-016X.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 26 Dec 2001
Last Updated on STN: 25 Feb 2002
ED Entered STN: 26 Dec 2001
Last Updated on STN: 25 Feb 2002

L81 ANSWER 13 OF 22 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:289147 BIOSIS Full-text
DOCUMENT NUMBER: PREV200200289147
TITLE: Immunotherapy for stomach cancer with the apoptosis-inducing human monoclonal antibody SC-1.
AUTHOR(S): **Vollmers, H. P.** [Reprint author]; Hensel, F. [Reprint author]; Braendlein, S. [Reprint author]; Timmermann, W.; Illert, B.; Wilhelm, M.; Reindl, L.; Thiede, A.; **Mueller-Hermelink, H. K.** [Reprint author]
CORPORATE SOURCE: Pathology, University Wuerzburg, Wuerzburg, Germany
SOURCE: European Journal of Cancer, (October, 2001) Vol. 37, No. Supplement 6, pp. S225. print.
Meeting Info.: 11th European Cancer Conference. Lisbon, Portugal. October 21-25, 2001.
CODEN: EJCAEL. ISSN: 0959-8049.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 15 May 2002
Last Updated on STN: 15 May 2002
ED Entered STN: 15 May 2002
Last Updated on STN: 15 May 2002

L81 ANSWER 14 OF 22 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2000:521286 BIOSIS Full-text
DOCUMENT NUMBER: PREV200000521286
TITLE: Immunotherapy for stomach carcinoma with the human monoclonal antibody SC-1 and the new DAF apoptosis pathway.
AUTHOR(S): **Vollmers, H. P.** [Reprint author]; Hensel, F. [Reprint author]; Krenn, V. [Reprint author]; Timmermann, W.; Illert, B.; Thiede, A.; **Mueller-Hermelink, H. K.** [Reprint author]
CORPORATE SOURCE: Inst. f. Pathologie, Universitaet Wuerzburg, Wuerzburg,

Germany
SOURCE: International Journal of Molecular Medicine, (2000) Vol. 6, No. Supplement 1, pp. S14. print.
Meeting Info.: Joint Meeting of the 5th World Congress on Advances in Oncology and the 3rd International Symposium on Molecular Medicine. Crete, Greece. October 19-21, 2000.
ISSN: 1107-3756.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 29 Nov 2000
Last Updated on STN: 11 Jan 2002
ED Entered STN: 29 Nov 2000
Last Updated on STN: 11 Jan 2002

L81 ANSWER 15 OF 22 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 1999:287837 BIOSIS Full-text
DOCUMENT NUMBER: PREV199900287837
TITLE: DAF/**CD55**, a new apoptosis surface receptor on stomach cancer cells defined by the human monoclonal antibody SC-1.
AUTHOR(S): Hermann, R. [Reprint author]; Hensel, F. [Reprint author]; Franke, A. [Reprint author]; Krenn, V. [Reprint author]; Geis, S. [Reprint author]; Abe, N. [Reprint author]; **Mueller-Hermelink, H. K.** [Reprint author]; **Vollmers, H. P.**
CORPORATE SOURCE: Inst. f. Pathologie, Universitaet Wuerzburg, Josef-Schneider-Str.2, 97080, Wuerzburg, Germany
SOURCE: European Journal of Cell Biology, (1999) Vol. 78, No. SUPPL. 49, pp. 25. print.
Meeting Info.: 23rd Annual Meeting of the German Society for Cell Biology. Rostock, Germany. March 14-18, 1999. German Society for Cell Biology.
CODEN: EJCBDN. ISSN: 0171-9335.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 5 Aug 1999
Last Updated on STN: 5 Aug 1999
ED Entered STN: 5 Aug 1999
Last Updated on STN: 5 Aug 1999

L81 ANSWER 16 OF 22 BIOTECHDS COPYRIGHT 2007 THE THOMSON CORP. on STN
ACCESSION NUMBER: 2005-16790 BIOTECHDS Full-text
TITLE: Novel purified SAM-6 antibodies capable of specifically binding to neoplastic cells, useful for diagnosing and treating proliferative diseases;
human antibody production against neoplastic cell via cell culture for use in disease therapy and diagnosis
AUTHOR: **VOLLMERS H; MUELLER-HERMELINK H K**
PATENT ASSIGNEE: VOLLMERS H; MUELLER-HERMELINK H K
PATENT INFO: EP 1531162 18 May 2005
APPLICATION INFO: EP 2003-26161 14 Nov 2003
PRIORITY INFO: EP 2003-26161 14 Nov 2003; EP 2003-26161 14 Nov 2003
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2005-357969 [37]
AN 2005-16790 BIOTECHDS Full-text
AB DERWENT ABSTRACT:

NOVELTY - A purified polypeptide (I) binding to neoplastic cells, having a sequence identical to a fully defined 96 amino acid (SEQ ID Number 1) and/or 110 amino acid (SEQ ID Number 3) sequence given in the specification, and specifically binding to BXPC-3 (ATCC Accession Number CRL-1687), 23132/87 (DSMZ Accession Number ACC 201), COLO-206F (DSMZ Accession Number ACC 21), COLO-699 (DSMZ Accession Number ACC 196) and LOU-NH91 (DSMZ Accession Number ACC 393) cells, is new.

DETAILED DESCRIPTION - A purified polypeptide (I) that binds to neoplastic cells, having an amino acid sequence substantially identical to a fully defined 96 amino acid (SEQ ID Number 1) and/or 110 amino acid (SEQ ID Number 3) sequence given in the specification, where (I) specifically binds to BXPC-3 (ATCC Accession Number CRL-1687), 23132 /87 (DSMZ Accession Number ACC 201), COLO-206F (DSMZ Accession Number ACC 21), COLO-699 (DSMZ Accession Number ACC 196), and LOU-NH91 (DSMZ Accession Number ACC 393) cells and not to non-neoplastic cells, where the neoplastic cell is a adenocarcinoma of the lung, squamous cell lung carcinoma, intestinal type gastric carcinoma, diffuse type gastric carcinoma, adenocarcinoma of the colon, adenocarcinoma of the prostate, squamous cell carcinoma of the esophagus, adenocarcinoma of the esophagus, adenocarcinoma of the esophagus lobular carcinoma of the breast, ductal carcinoma of the breast, adenocarcinoma of the pancreas, adenocarcinoma of the ovary and adenocarcinoma of the uterus cell, where (I) is expressed by the hybridoma cell line, which was deposited at the DSMZ (Deutsche Sammlung von Mikrorganismen and Zellkulturen GmbH in Braunschweig Germany) as SAM-6 on the 7th of November 2003. INDEPENDENT CLAIMS are also included for the following: (1) a complementarity-determining region (CDR) or its functional fragment comprising the amino acid sequence substantially identical to the amino acid sequence Ser-Gly-Asp-Lys-Leu-Gly-Asp-Lys-Tyr-Ala-Cys (CDR1) or Gln-Asp-Ser-Lys-Arg-Pro-Ser (CDR2) Or Gln-Ala-Trp-Asp-Ser-Ser-Ile-Val-Val (CDR3) of SEQ ID Number 1 and/or Ser-Tyr-Ala-Met-His (CDR1) or Val-Ile-Ser-Tyr-Asp-Gly-Ser-Asn-Lys-Tyr-Tyr- Ala-Asp-Ser-Val-Lys-Gly (CDR2) or Asp-Arg-Leu-Ala-Gly-Lys-Thr-Phe-Asp-Tyr of SEQ ID Number 3; (2) a cell (II) that expressing (I), or a polypeptide having a sequence that is substantially identical to the amino acid sequence of SEQ ID Number 1 and/or SEQ ID Number 3; (3) a hybridoma cell line, which was deposited at the DSMZ; (4) generating (II), involves contacting lymphocytes with a heteromyeloma cell line under conditions that result in the fusion of a lymphocyte with a heteromyeloma cell, the fusion resulting in a hybridoma, determining whether the hybridoma produces a polypeptide that inhibits proliferation in a neoplastic cell to which it binds, but does not inhibit proliferation in a non-neoplastic cell, or determining whether the hybridoma produces a polypeptide that induces intracellular accumulation of lipids in a neoplastic cell to which it binds, but does not induce intracellular accumulation of lipids in a non-neoplastic cell, or determining whether the hybridoma produces a polypeptide that induces apoptosis of a neoplastic cell to which it binds, but does not induce apoptosis of a non-neoplastic cell, and determining whether the hybridoma produces a polypeptide that specifically binds to BXPC-2 (ATCC Accession number CRL-1687), 23132/87 (DSMZ Accession number ACC 201), COLO-206F (DSMZ Accession number ACC 21), COLO-699 (DSMZ Accession number ACC 196), and LOU-NH91 (DSMZ Accession number ACC 393) cells and not to non-neoplastic cells; (5) a diagnostic agent comprising (I); (6) an isolated nucleic acid molecule (III) comprising a fully defined 288 nucleotide (SEQ ID Number 2) or 330 nucleotide (SEQ ID Number 4) sequence given in the specification; (7) a vector (IV) comprising (III); and (8) a cell comprising (IV). BIOTECHNOLOGY - Preferred Polypeptide: (I) inhibits cell proliferation when bound to a neoplastic cell, but does not inhibit cell proliferation of a non-neoplastic cell. (I) induces the intracellular accumulation of lipids when bound to a neoplastic cell, but does not induce the intracellular accumulation of lipids in a non-neoplastic cell. (I) induces apoptosis of a neoplastic cell to which it binds, but does not induce apoptosis of a non-neoplastic cell. (I) comprises an antibody or its

functional fragment, which is chosen from VL, VH, FV, FC, Fab, Fab' and F(ab')2. (I) has an amino acid sequence of the variable region of the light chain (VL) substantially identical to SEQ ID Number 1 or SEQ ID Number 2, and/or an amino acid sequence of the variable region of the heavy chain (VH) substantially identical to SEQ ID Number 3 or SEQ ID Number 4. The functional fragment comprises a fragment of the sequence of SEQ ID Number 1 and SEQ ID Number 3. The functional fragment comprises a fragment that is substantially identical to the sequence SEQ ID Number 1 and/or SEQ ID Number 3. (I) comprises nucleic acid sequences that are substantially identically to nucleotides 67-99 (CDR1), 145-165 (CDR2) and 262-288 (CDR3) of SEQ ID Number 2. (I) comprises nucleic acid sequences that are substantially identically to nucleotides 91-105 (CDR1), 148-198 (CDR2) and 295-330 (CDR3) of SEQ ID Number 4. (I) is a monoclonal antibody, preferably a human monoclonal antibody.
ACTIVITY - Cytostatic.
MECHANISM OF ACTION - Inducer of apoptosis; Inhibitor of cell proliferation; Inducer of intracellular accumulation of lipids (claimed). Cell death detection enzyme linked immunosorbent assay (ELISA) was performed using 1x104 tumor cells (BXPC-3, 23132/87, RPMI-2650 and HNEpC-c) incubated with SAM-6 antibodies and control antibodies. The level of the antibody-induced apoptosis was calculated based on the color intensity. The results show that SAM-6 induced apoptosis in carcinoma cells after 48 hours of incubation.
USE - (I) is useful for diagnosing a neoplasm in a mammal, which involves contacting a cell or tissue of the mammal with (I), and detecting whether the purified polypeptide binds to the cell or tissue sample, where binding of (I) to the cell or tissue sample is indicative of the mammal having a neoplasm. The mammal is human. The neoplasm is a adenocarcinoma of the lung, squamous cell lung carcinoma, intestinal type gastric carcinoma, diffuse type gastric carcinoma, adenocarcinoma of the colon, adenocarcinoma of the prostate, squamous cell carcinoma of the esophagus, adenocarcinoma of the esophagus, adenocarcinoma of the esophagus lobular carcinoma of the breast, ductal carcinoma of the breast, adenocarcinoma of the pancreas, adenocarcinoma of the ovary and adenocarcinoma of the uterus cell. (I) is an antibody conjugated to a detectable agent chosen from radionuclide, fluorescent marker, enzyme, cytotoxin, cytokine and growth inhibitor. (I) is conjugated to a protein purification tag, which is cleavable. (I) is also useful for treating the proliferative disorder in a mammal, which involves contacting a cell or tissue sample with (I), where binding of (I) to the cell or tissue sample results in a reduction in proliferation, intracellular accumulation of lipids, and induction of apoptosis of the cell or of a cell in the tissue sample, where (I) is conjugated to the detectable agent, which is capable of inhibiting cell proliferation of the cell or tissue sample. (I) along with carrier is useful for the production of medicament that inhibits cell proliferation, induces the intracellular accumulation of lipids or that induces apoptosis (claimed). ADMINISTRATION - (I) is administered by intramuscular, intravenous, intraperitoneal, intravesicular, intraarticular, intralesional, or subcutaneous route at dosage of 0.1-50 mg/kg body weight.
EXAMPLE - No relevant example is given. (47 pages)

L81 ANSWER 17 OF 22 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2006397391 EMBASE Full-text
TITLE: Natural IgM antibodies: The orphaned molecules in immune surveillance.
AUTHOR: **Vollmers H.P.**; Brandlein S.
CORPORATE SOURCE: H.P. Vollmers, Institute for Pathology, University Wurzburg, Josef-Schneider-Str. 2, D-97080 Wurzburg, Germany. peter.vollmers@mail.uni-wuerzburg.de
SOURCE: Advanced Drug Delivery Reviews, (7 Aug 2006) Vol. 58, No. 5-6, pp. 755-765. .

Refs: 98
ISSN: 0169-409X CODEN: ADDREP
PUBLISHER IDENT.: S 0169-409X(06)00093-7
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 016 Cancer
026 Immunology, Serology and Transplantation
037 Drug Literature Index
039 Pharmacy
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 1 Sep 2006
Last Updated on STN: 1 Sep 2006
ED Entered STN: 1 Sep 2006
Last Updated on STN: 1 Sep 2006

AB Natural IgM antibodies are typical victims of prejudices which originated in the mid 80 s. Over the years, these molecules were considered as the pariahs among the immune competent molecules and their characteristic properties, like low affinity, cross-reactivity and pentameric structure, were assessed as useless, difficult, nebulous, etc. Today, mainly based on a few scientists' persistent work and the key discoveries on innate immune recognition, natural IgM antibodies are "back on stage". Their role in the immune response against bacteria, viruses, fungi and possibly modified self-components as well as in therapy and diagnosis of malignancies is accepted. All the so far negatively judged features are seen in a different light, e.g. low affinity seems to be good for function and does not exclude specificity, and cross-reactivity is no longer judged as unspecific, but instead as a very economic way of immune recognition. And at last, with the use of natural IgM antibodies, a new field of tumor-specific targets has been encountered, the carbo-neo-epitopes. Therefore, by having learned from nature, the renaissance of natural IgM antibodies opens a new area of cancer therapeutics and diagnostics. .COPYRGT. 2006 Elsevier B.V. All rights reserved.

L81 ANSWER 18 OF 22 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2005300597 EMBASE Full-text
TITLE: The "early birds": Natural IgM antibodies and immune surveillance.
AUTHOR: **Vollmers H.P.**; Brandlein S.
CORPORATE SOURCE: Dr. H.P. Vollmers, Institute of Pathology, University of Wurzburg, Josef-Schneider-Str. 2, D-97080 Wurzburg, Germany. peter.vollmers@mail.uni-wuerzburg.de
SOURCE: Histology and Histopathology, (2005) Vol. 20, No. 3, pp. 927-937. .
Refs: 71
ISSN: 0213-3911 CODEN: HIHIES
COUNTRY: Spain
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 016 Cancer
022 Human Genetics
026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 21 Jul 2005
Last Updated on STN: 21 Jul 2005
ED Entered STN: 21 Jul 2005
Last Updated on STN: 21 Jul 2005

AB Precancerous epithelial lesions are sites of uncontrolled cellular proliferation generated by irreversible genetic alterations. Not all of those lesions progress to invasive cancer, some may even regress, but the early detection of abnormal cells can be crucial for patient survival. Immune surveillance mechanisms are responsible for the removal of transformed cells and antibodies play an important role in these immune processes. In the past, analysis of the immunoglobuline repertoire has focused mainly on xenoimmunizations or the investigation of cancer patient immunity. The human hybridoma technology (Trioma technique) offers the unique possibility to study the humoral immunity of healthy people. Using this technique a series of tumor-binding antibodies could be isolated which all have several features in common: they are germ-line coded IgM antibodies, they predominantly bind to carbohydrates on post-transcriptionally modified antigens, they induce apoptosis and, most importantly, they detect not only malignant cells but also precursor stages. These data demonstrate that the body has a comprehensive defense system against malignant cells based on the production of natural antibodies.

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ACCESSION NUMBER: 2005191155 EMBASE Full-text
TITLE: Death by stress: Natural IgM-induced apoptosis.
AUTHOR: **Vollmers H.P.**; Brandlein S.
CORPORATE SOURCE: Dr. H.P. Vollmers, Institut fur Pathologie, Universitat Wurzburg, Josef-Schneider Str. 2, D-97080 Wurzburg, Germany. peter.vollmers@mail.uni-wuerzburg.de
SOURCE: Methods and Findings in Experimental and Clinical Pharmacology, (2005) Vol. 27, No. 3, pp. 185-191. .
Refs: 77
ISSN: 0379-0355 CODEN: MFEPDX
COUNTRY: Spain
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 016 Cancer
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 26 May 2005
Last Updated on STN: 26 May 2005
ED Entered STN: 26 May 2005
Last Updated on STN: 26 May 2005
AB Stress kills and hence should be avoided. On the other hand, stress induction can be used to remove malignant cells by inducing cellular suicide. Natural IgM antibodies act as first-line defense in immune surveillance. These antibodies selectively kill aberrant cells by using different apoptotic stress mechanisms. They can be isolated from patients but also from healthy donors by using the human hybridoma technology. They are components of the innate immunity, and, on the basis of specific screening methods, should also be detectable in any other individual. The three tumor-specific, apoptosis-inducing natural IgM antibodies described in this review are good examples for stress-induced apoptosis and nature's resourceful ways to fight malignant growth. .COPYRGT. 2005 Prous Science. All rights reserved.

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ACCESSION NUMBER: 97252306 EMBASE Full-text
DOCUMENT NUMBER: 1997252306
TITLE: Immunopathological observations after xenogeneic liver perfusions using donor pigs transgenic for human decay-accelerating factor.

AUTHOR: Pascher A.; Poehlein C.; Storck M.; Prestel R.; **Mueller-Hoecker J.**; White D.J.G.; Abendroth D.; Hammer C.

CORPORATE SOURCE: Prof. C. Hammer, Institute for Surgical Research, Klinikum Grosshadern, LMU Munich, Marchioninistr. 15, 81366 Munich, Germany

SOURCE: Transplantation, (1997) Vol. 64, No. 3, pp. 384-391. .
Refs: 23
ISSN: 0041-1337 CODEN: TRPLAU

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 005 General Pathology and Pathological Anatomy
009 Surgery
048 Gastroenterology

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 4 Sep 1997
Last Updated on STN: 4 Sep 1997

ED Entered STN: 4 Sep 1997
Last Updated on STN: 4 Sep 1997

AB Background. Donor pigs transgenic for human decay-accelerating factor (hDAF) were used in a xenogeneic ex vivo liver perfusion model to study the effect of this modification on the development of hyperacute rejection. Methods. Three transgenic pigs were hepatectomized after hypothermic portal and transaortal gravity perfusion. Livers from six nontransgenic pigs served as controls. All livers were perfused for 3 hr with human blood from two donors diluted to a hematocrit of 30%. Particular importance was placed on the use of an optimal perfusion technique incorporating the floating suspension of the organs in a waterbath and intermittent external pressurization. Biochemical, physiological, and immunological parameters were assessed. Tissue specimens taken before and after perfusion were analyzed using routine histology, electron microscopy, and immunohistology. Results. Complement activation was more pronounced in the control group. AP50 and CH50 values fell to about 60% of the initial levels in control experiments, whereas they remained at 80% of the initial levels during perfusion of hDAF livers. After 180 min, pig tumor necrosis factor α levels were 7862±1645 pg/ml for unmodified livers and 2830±734 pg/ml in the hDAF group. Human tumor necrosis factor a levels were similar in both groups. Control livers showed marked morphological alterations and distinct deposition of complement factors, whereas livers expressing hDAF showed no signs of hepatocellular necrosis and almost no complement deposition beyond C3 activation. Conclusions. These results confirm that the transgenic expression of the human complement regulatory protein hDAF reduces complement activation and prevents hyperacute rejection in a xenogeneic liver perfusion model over the 3-hr evaluation period used in this study.

L81 ANSWER 21 OF 22 EMBASE

ACCESSION NUMBER: 96138210 EMBASE Full-text

DOCUMENT NUMBER: 1996138210

TITLE: Human decay accelerating factor expressed on endothelial cells of transgenic pigs affects complement activation in an ex vivo liver perfusion model.

AUTHOR: Pascher A.; Poehlein C.; Storck M.; Abendroth D.; **Mueller-Hoecker J.**; Young V.K.; Koenig W.; White D.J.G.; Hammer C.

CORPORATE SOURCE: Klinikum Grosshadern, Institute for Surgical Research, Marchioninistr 15,81366 Muenchen, Germany

SOURCE: Transplantation Proceedings, (1996) Vol. 28, No. 2, pp. 754-755. .

ISSN: 0041-1345 CODEN: TRPPA8
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 026 Immunology, Serology and Transplantation
048 Gastroenterology
LANGUAGE: English
ENTRY DATE: Entered STN: 29 May 1996
Last Updated on STN: 29 May 1996
ED Entered STN: 29 May 1996
Last Updated on STN: 29 May 1996
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L81 ANSWER 22 OF 22 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 96351176 EMBASE Full-text
DOCUMENT NUMBER: 1996351176
TITLE: Expression of human decay accelerating factor (hDAF) in transgenic pigs regulates complement activation during ex vivo liver perfusion - Immunopathological findings.
AUTHOR: Pascher A.; Poehlein Ch.; Storck M.; Abendroth D.; **Mueller-Hoecker J.**; Koenig W.; Young V.K.; White D.J.G.; Hammer C.
CORPORATE SOURCE: Institute for Surgical Research, Klinikum Grosshadern, LMU Munich, Marchioninistrasse 15,D-81366 Munich, Germany
SOURCE: Transplant International, (1996) Vol. 9, No. SUPPL. 1, pp. S385-S387. .
ISSN: 0934-0874 CODEN: TRINE5
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 009 Surgery
026 Immunology, Serology and Transplantation
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1996
Last Updated on STN: 10 Dec 1996
ED Entered STN: 10 Dec 1996
Last Updated on STN: 10 Dec 1996
AB Ex vivo perfusions of human decay accelerating factor-expressing transgenic (n = 3), and nontransgenic (n = 6) porcine livers with human blood revealed a higher degree of organ damage in nontransgenic pig livers. Transgenic livers were protected from immunohistologically detectable complement deposition, despite corresponding IgM and IgG deposits in both groups. Complement activation and consumption of C3 and C4 turned out to be lower in transgenic pig livers. In contrast to livers of normal landrace pigs, livers from genetically manipulated pigs showed no morphological alterations after perfusion.

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TEXT SEARCH
=>
=> => fil medl; d que l32; d que l35; d que l33
FILE 'MEDLINE' ENTERED AT 17:01:51 ON 17 JAN 2007

 FILE LAST UPDATED: 16 Jan 2007 (20070116/UP).  FILE COVERS 1950 TO DATE.

  All regular MEDLINE updates from November 15 to December 16 have been
  added to MEDLINE, along with 2007 Medical Subject Headings (MeSH(R))
  and 2007 tree numbers.

  The annual reload will be available in early 2007.

  This file contains CAS Registry Numbers for easy and accurate
  substance identification.


L26         1166 SEA FILE=MEDLINE ABB=ON  ANTIGENS, CD55/CT
L29           24 SEA FILE=MEDLINE ABB=ON  23132
L32            0 SEA FILE=MEDLINE ABB=ON  L26 AND L29



L26         1166 SEA FILE=MEDLINE ABB=ON  ANTIGENS, CD55/CT
L27        53324 SEA FILE=MEDLINE ABB=ON  GLYCOPROTEINS/CT
L28       203676 SEA FILE=MEDLINE ABB=ON  ADENOCARCINOMA+NT/CT
L35            0 SEA FILE=MEDLINE ABB=ON  L26 AND L28 AND L27



L26         1166 SEA FILE=MEDLINE ABB=ON  ANTIGENS, CD55/CT
L28       203676 SEA FILE=MEDLINE ABB=ON  ADENOCARCINOMA+NT/CT
L30       393194 SEA FILE=MEDLINE ABB=ON  CELL LINE+NT/CT
L33            5 SEA FILE=MEDLINE ABB=ON  L26 AND L28 AND L30


=> s l33 not l25

L82            5 L33 NOT L25

=> fil embase; d que l47; d que l49; d que l55

FILE 'EMBASE' ENTERED AT 17:01:52 ON 17 JAN 2007
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 FILE COVERS 1974 TO 17 Jan 2007 (20070117/ED)

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 This file contains CAS Registry Numbers for easy and accurate
 substance identification.


L39         1317 SEA FILE=EMBASE ABB=ON  DECAY ACCELERATING FACTOR/CT
L41        16843 SEA FILE=EMBASE ABB=ON  ADENOCARCINOMA/CT
L47            3 SEA FILE=EMBASE ABB=ON  L39 AND L41
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L39          1317 SEA FILE=EMBASE ABB=ON  DECAY ACCELERATING FACTOR/CT
L42         42527 SEA FILE=EMBASE ABB=ON  CELL LINE/CT
L43          9850 SEA FILE=EMBASE ABB=ON  TUMOR CELL LINE/CT
L45         27881 SEA FILE=EMBASE ABB=ON  GLYCOPROTEIN/CT
L49             0 SEA FILE=EMBASE ABB=ON  L39 AND (L42 OR L43) AND L45



L39          1317 SEA FILE=EMBASE ABB=ON  DECAY ACCELERATING FACTOR/CT
L40             5 SEA FILE=EMBASE ABB=ON  23132?
L55             0 SEA FILE=EMBASE ABB=ON  L39 AND L40


=> s l47 not l46

L83             3 L47 NOT L46

=> fil wpix; d que l60; d que l61; d que l66

FILE 'WPIX' ENTERED AT 17:01:54 ON 17 JAN 2007
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FILE LAST UPDATED:                          15 JAN 2007   <20070115/UP>
MOST RECENT THOMSON SCIENTIFIC UPDATE:     200704      <200704/DW>
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    EPI,  please go to:
    http://scientific.thomson.com/dwpi-manualcoderevision <<<
'BI ABEX' IS DEFAULT SEARCH FIELD FOR 'WPIX' FILE


L52           119 SEA FILE=WPIX ABB=ON  CD55/BI,ABEX OR CD 55/BI,ABEX OR DECAY
                  ACCELERATING FACTOR/BI,ABEX
L58          3007 SEA FILE=WPIX ABB=ON  ADENOCARCINOMA#/BI,ABEX OR ADENO/BI,ABEX(
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L60           4 SEA FILE=WPIX ABB=ON  L52 AND L58


L52         119 SEA FILE=WPIX ABB=ON  CD55/BI,ABEX OR CD 55/BI,ABEX OR DECAY
                ACCELERATING FACTOR/BI,ABEX
L59           5 SEA FILE=WPIX ABB=ON  23132?/BI,ABEX
L61           1 SEA FILE=WPIX ABB=ON  L52 AND L59



L52         119 SEA FILE=WPIX ABB=ON  CD55/BI,ABEX OR CD 55/BI,ABEX OR DECAY
                ACCELERATING FACTOR/BI,ABEX
L56        3675 SEA FILE=WPIX ABB=ON  B04-N06/MC OR C04-N06/MC = Glyco proteins
L57        6999 SEA FILE=WPIX ABB=ON  GLYCOPROTEIN#/BI,ABEX OR GLYCO PROTEIN#/B
                I,ABEX
L62          20 SEA FILE=WPIX ABB=ON  L52 AND (L56 OR L57)
L63      220303 SEA FILE=WPIX ABB=ON  MW/BI,ABEX OR MOL?/BI,ABEX(W)WEIGHT/BI,AB
                EX OR KDA/BI,ABEX OR DALTON#/BI,ABEX OR KILODALTON#/BI,ABEX OR
                KD/BI,ABEX
L65      132265 SEA FILE=WPIX ABB=ON  82/BI,ABEX OR 82000/BI,ABEX
L66           2 SEA FILE=WPIX ABB=ON  (L65 OR L63) AND L62


=> s l60,l61,l66 not l53

L84           4 (L60 OR L61 OR L66) NOT L53

=> fil capl; d que l10; d que l17; d que l22; d que l8

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L4          362 SEA FILE=CAPLUS ABB=ON  CD55/OBI OR CD 55/OBI
L6        21213 SEA FILE=CAPLUS ABB=ON  CARCINOMA#/OBI(L)ADENO/OBI OR ADENOCARC
                INOMA#/OBI
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L10           6 SEA FILE=CAPLUS ABB=ON  L4 AND L6


L6        21213 SEA FILE=CAPLUS ABB=ON  CARCINOMA#/OBI(L)ADENO/OBI OR ADENOCARC
                INOMA#/OBI
L11           1 SEA FILE=REGISTRY ABB=ON  99085-47-9
L12         967 SEA FILE=CAPLUS ABB=ON  DECAY-ACCELERATING FACTOR/OBI
L13        1086 SEA FILE=CAPLUS ABB=ON  L11
L17           8 SEA FILE=CAPLUS ABB=ON  L6 (L)(L12 OR L13)



L6        21213 SEA FILE=CAPLUS ABB=ON  CARCINOMA#/OBI(L)ADENO/OBI OR ADENOCARC
                INOMA#/OBI
L11           1 SEA FILE=REGISTRY ABB=ON  99085-47-9
L12         967 SEA FILE=CAPLUS ABB=ON  DECAY-ACCELERATING FACTOR/OBI
L13        1086 SEA FILE=CAPLUS ABB=ON  L11
L20      114772 SEA FILE=CAPLUS ABB=ON  GLYCOPROTEIN#/OBI
L21         177 SEA FILE=CAPLUS ABB=ON  PROTEIN#/OBI(L)GLYCO/OBI
L22           6 SEA FILE=CAPLUS ABB=ON  L6 AND (L20 OR L21) AND (L12 OR L13)



L4          362 SEA FILE=CAPLUS ABB=ON  CD55/OBI OR CD 55/OBI
L7            4 SEA FILE=CAPLUS ABB=ON  23132/OBI
L8            0 SEA FILE=CAPLUS ABB=ON  L4 AND L7


=> s l10,l17,l22 not l80

L85          14 (L10 OR L17 OR L22) NOT L80

=> fil jic pascal biotechno biosis esbio biotechds lifesci confsci dissabs bioeng
scisearch

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=> d que l77; d que l79; s l77,l79 not l76

L69 7655 SEA CD55 OR CD 55 OR DECAY ACCELERATING FACTOR
L71 51 SEA 23132?
L77 1 SEA L69 AND L71

L69 7655 SEA CD55 OR CD 55 OR DECAY ACCELERATING FACTOR
L70 546531 SEA GLYCOPROTEIN# OR GLYCO PROTEIN#
L72 225456 SEA ADENOCARCINOMA# OR ADENO(A) CARCINOMA#
L73 1412828 SEA MW OR MOL?(W) WEIGHT OR KDA OR DALTON# OR KILODALTON# OR KD OR 82 OR 82000
L78 103 SEA L69 AND L72
L79 18 SEA L78 AND (L70 OR L73)

L86 15 (L77 OR L79) NOT L76

=> => dup rem l82,l85,l84,l83,l86
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PROCESSING COMPLETED FOR L82
PROCESSING COMPLETED FOR L85
PROCESSING COMPLETED FOR L84
PROCESSING COMPLETED FOR L83
PROCESSING COMPLETED FOR L86
L87 28 DUP REM L82 L85 L84 L83 L86 (13 DUPLICATES REMOVED)
ANSWERS '1-5' FROM FILE MEDLINE
ANSWERS '6-18' FROM FILE CAPLUS
ANSWERS '19-20' FROM FILE WPIX
ANSWERS '21-22' FROM FILE EMBASE
ANSWER '23' FROM FILE JICST-EPLUS
ANSWER '24' FROM FILE PASCAL
ANSWER '25' FROM FILE BIOSIS
ANSWERS '26-28' FROM FILE SCISEARCH

=> d iall 1-5; d ibib ed abs hitind 6-18; d iall abeq tech 19-20; d iall 21-28; fil hom

L87 ANSWER 1 OF 28 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2001653278 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 11706063
TITLE: Regulation of the new coexpressed CD55 (decay-accelerating factor) receptor on stomach carcinoma cells involved in antibody SC-1-induced apoptosis.
AUTHOR: Hensel F; Hermann R; Brandlein S; Krenn V; Schmausser B; Geis S; Muller-Hermelink H K; Vollmers H P
CORPORATE SOURCE: Institute for PathologyUniversity of Wurzburg, Wurzburg, Germany.
SOURCE: Laboratory investigation; a journal of technical methods and pathology, (2001 Nov) Vol. 81, No. 11, pp. 1553-63. Journal code: 0376617. ISSN: 0023-6837.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 14 Nov 2001
Last Updated on STN: 23 Jan 2002
Entered Medline: 7 Dec 2001
ABSTRACT:
The human monoclonal antibody SC-1 was isolated from a patient with a diffuse-type adenocarcinoma of the stomach using somatic cell hybridization. The immunoglobulin (Ig)M antibody reacts specifically with diffuse- (70%) and intestinal-type (25%) gastric adenocarcinoma and induces apoptosis in vitro and in vivo. When used in clinical trials with stomach carcinoma patients, significant apoptotic and regressive effects in primary tumors have been observed with the antibody SC-1. The SC-1 receptor is a new 82 kd membrane-bound isoform of glycosylphosphatidylinositol (GPI)-linked CD55 (decay-accelerating factor, DAF). CD55 is known to protect cells from lysis through autologous complement and is coexpressed with the ubiquitously distributed 70 kd isoform. The SC-1-specific CD55 isoform is up-regulated shortly after antibody binding, followed by an internalization of the antibody/receptor-complex, whereas the membranous expression of wild-type CD55 remains unchanged. The apoptotic process is marked by cleavage of cytokeratin

18, indicating the involvement of caspase-6 in the apoptotic process. In contrast to other apoptotic pathways, no cleavage of poly(ADP-ribose)polymerase (PARP) is not observed. The expression of the cell-cycle regulator c-myc becomes up-regulated, whereas expression of topoisomerase IIalpha is down-regulated. Induction of apoptosis leads to an increase in the internal Ca(2+) concentration, which is not necessary for the apoptotic process but for the transport of newly synthesized SC-1-specific CD55 isoform to the membrane.

CONTROLLED TERM:
***Adenocarcinoma**
*Antibodies, Monoclonal: PK, pharmacokinetics
Antigens, CD55: AN, analysis
***Antigens, CD55: BI, biosynthesis**
Antigens, CD55: IM, immunology
*Antineoplastic Agents: PK, pharmacokinetics
*Apoptosis: IM, immunology
Calcium: ME, metabolism
Caspases: AI, antagonists & inhibitors
Cell Membrane: PH, physiology
Cytoplasm: PH, physiology
Flow Cytometry
Hela Cells
Humans
Keratin: ME, metabolism
Poly(ADP-ribose) Polymerases: ME, metabolism
Research Support, Non-U.S. Gov't
*Stomach Neoplasms

CAS REGISTRY NO.: 68238-35-7 (Keratin); 7440-70-2 (Calcium)

CHEMICAL NAME: 0 (Antibodies, Monoclonal); 0 (Antigens, CD55); 0 (Antineoplastic Agents); 0 (SC-1 monoclonal antibody); EC 2.4.2.30 (Poly(ADP-ribose) Polymerases); EC 3.4.22.- (Caspases); EC 3.4.22.- (caspase 6); EC 3.4.22.- (caspase-3)

L87 ANSWER 2 OF 28 MEDLINE on STN DUPLICATE 7

ACCESSION NUMBER: 95357637 MEDLINE Full-text

DOCUMENT NUMBER: PubMed ID: 7543216

TITLE: Augmented lung adenocarcinoma cytotoxicity by the combination of a genetically modified anti-Lewis Y antibody and antibodies to complement regulatory proteins.

AUTHOR: Azuma A; Yamano Y; Yoshimura A; Hibino T; Nishida T; Yagita H; Okumura K; Seya T; Kannagi R; Shibuya M; +

CORPORATE SOURCE: Fourth Department of Internal Medicine, Nippon Medical School, Tokyo, Japan.

SOURCE: Scandinavian journal of immunology, (1995 Aug) Vol. 42, No. 2, pp. 202-8.
Journal code: 0323767. ISSN: 0300-9475.

PUB. COUNTRY: ENGLAND: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199509

ENTRY DATE: Entered STN: 21 Sep 1995
Last Updated on STN: 29 Jan 1996
Entered Medline: 7 Sep 1995

ABSTRACT:
Complement-dependent cytotoxicity (CDC) mediated by a chimeric anti-Lewis Y monoclonal antibody (cH18A; human IgG1) was investigated in this study. Human lung adenocarcinoma cell lines (PC7, PC9, and PC14) were used as the target cells. PC7 and PC9 cells, expressed Lewis Y antigen and were lysed by cH18A as effectively as by the parent mouse anti-Lewis Y antibodies (mH18A) in a concentration-dependent manner. PC14 cells did not express Lewis Y antigen and

were not lysed by either cH18A or mH18A. cH18A mediated CDC activity against PC7 and PC9 cells was enhanced by the combined use of monoclonal antibodies directed against CD46(MCP), CD55(DAF), and CD59. These molecules are complement-regulatory proteins which protect host cells from CDC. PC7 and PC9 cells, showed high levels of surface expression of these proteins, PC7 cells were more susceptible to cH18A-mediated CDC than PC9 cells. Use of multiple blocking antibodies to the complement-regulatory proteins produced more enhancement of cH18A-mediated CDC than a single antibody. Moreover, expression of CD55 and CD59 by PC7 and PC9 cells was decreased after treatment with PI-PLC, resulting in increased susceptibility to cH18A-mediated CDC. Although the reason is unknown, PC7 cells became more susceptible to CDC than PC9 cells after PI-PLC treatment even in the absence of cH18A. These data suggest that chimeric monoclonal antibodies can be used to induce CDC against lung adenocarcinoma, and that such CDC is potentiated by a variety of antibodies blocking compliment-regulatory proteins on the tumour cell surface.

CONTROLLED TERM: ***Adenocarcinoma: IM, immunology**
Animals
Antibodies, Monoclonal: IM, immunology
*Antibody-Dependent Cell Cytotoxicity
*Antigens, CD: IM, immunology
Antigens, CD46
Antigens, CD55
Antigens, CD59
Cell Line, Transformed
Humans
*Lewis Blood-Group System: IM, immunology
*Lung Neoplasms: IM, immunology
*Membrane Glycoproteins: IM, immunology
Mice

CHEMICAL NAME: 0 (Antibodies, Monoclonal); 0 (Antigens, CD); 0 (Antigens, CD46); 0 (Antigens, CD55); 0 (Antigens, CD59); 0 (CD46 protein, human); 0 (Lewis Blood-Group System); 0 (Lewis Y antigen); 0 (Mcp protein, mouse); 0 (Membrane Glycoproteins)

L87 ANSWER 3 OF 28 MEDLINE on STN
ACCESSION NUMBER: 2006235254 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 16640659
TITLE: Minimal residual disease in ovarian cancer as a target for complement-mediated mAb immunotherapy.
AUTHOR: Bjorge L; Stoiber H; Dierich M P; Meri S
CORPORATE SOURCE: Department of Obstetrics and Gynecology, Institute of Clinical Medicine, Haukeland University Hospital, Bergen, Norway.. line.bjorge@gades.uib.no
SOURCE: Scandinavian journal of immunology, (2006 May) Vol. 63, No. 5, pp. 355-64.
Journal code: 0323767. ISSN: 0300-9475.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200606
ENTRY DATE: Entered STN: 28 Apr 2006
Last Updated on STN: 10 Jun 2006
Entered Medline: 9 Jun 2006

ABSTRACT:
Ovarian cancer is potentially well suited for local monoclonal antibody (mAb) immunotherapy, because it remains within the peritoneal cavity for a long period of time before giving rise to distant metastases. At the stage of minimal residual disease, the cells appear to be in a state of dormancy (G(0))

or at least have lower rates of tumour cell proliferation. They should be a promising target for immunotherapy. Here we first examined the cell-cycle expression of CD59 and decay-accelerating factor (DAF; CD55) on four different ovarian carcinoma cell lines, using simultaneous flow cytometric analysis of DNA content or the cell-cycle-specific nuclear proliferation protein Ki67 and CD59 or DAF surface expression. We found that CD59 and DAF are stably expressed throughout the cell cycle. The polyvalent approach to target-independent antigens to improve the efficiency of mAb complement (C)-mediated damages was promising, and tumour cells become sensitive to C damage, when incubated with cross-linked mAb against different tumour-associated antigens. Although, such immune complex-mediated C activation was rather ineffective in killing the cells, it could be potentiated by the addition of blocking mAb against CD59 and DAF. Our results suggest that the activities of intrinsic C regulators must be neutralized to make minimal residual disease a promising target for antibody therapy.

CONTROLLED TERM: Check Tags: Female
***Adenocarcinoma: DT, drug therapy**
Adenocarcinoma: IM, immunology
*Antibodies, Blocking: TU, therapeutic use
*Antibodies, Monoclonal: TU, therapeutic use
Antigens, CD55: AN, analysis
***Antigens, CD55: DE, drug effects**
Antigens, CD55: ME, metabolism
Antigens, Neoplasm: AN, analysis
Cell Cycle
Cell Line, Tumor
*Complement System Proteins: IM, immunology
DNA, Neoplasm: AN, analysis
Flow Cytometry
Humans
Immunotherapy
Ki-67 Antigen: AN, analysis
Ki-67 Antigen: ME, metabolism
Neoplasm, Residual
*Ovarian Neoplasms: DT, drug therapy
Ovarian Neoplasms: IM, immunology
Research Support, Non-U.S. Gov't

CAS REGISTRY NO.: 9007-36-7 (Complement System Proteins)

CHEMICAL NAME: 0 (Antibodies, Blocking); 0 (Antibodies, Monoclonal); 0 (Antigens, CD55); 0 (Antigens, Neoplasm); 0 (DNA, Neoplasm); 0 (Ki-67 Antigen)

L87 ANSWER 4 OF 28 MEDLINE on STN

ACCESSION NUMBER: 2000511822 MEDLINE Full-text

DOCUMENT NUMBER: PubMed ID: 11069077

TITLE: A therapeutic human anti-idiotypic antibody mimics CD55 in three distinct regions.

AUTHOR: Spendlove L; Li L; Potter V; Christiansen D; Loveland B E; Durrant L G

CORPORATE SOURCE: CRC Academic Unit of Clinical Oncology, University of Nottingham, City Hospital, GB..
Ian.Spendlove@Nottingham.ac.uk

SOURCE: European journal of immunology, (2000 Oct) Vol. 30, No. 10, pp. 2944-53.
Journal code: 1273201. ISSN: 0014-2980.

PUB. COUNTRY: GERMANY: Germany, Federal Republic of

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200012

ENTRY DATE: Entered STN: 22 Mar 2001
Last Updated on STN: 22 Mar 2001
Entered Medline: 7 Dec 2000

ABSTRACT:
The human anti-idiotypic antibody 105AD7 was isolated from a colorectal cancer patient receiving the anti-tumor antibody 791T/36 for radioimmuno-scintigraphy of liver metastases. We have mapped the binding site of 791T/36 to the first two small consensus repeat (SCR) domains of the complement regulatory protein (CD55) that is overexpressed by a wide range of solid tumors. Cloning of both antigen and anti-idiotype has identified the molecular basis of their mimicry. Amino acid homology has been identified between three complementarity-determining regions of 105AD7 and three regions of CD55 within the first two SCR domains. 791T/36 and anti-anti-idiotypic (Ab3) polyclonal antibodies raised against 105AD7 showed specific binding to these peptides. The antibodies were also found to bind synergistically to combinations of these peptides, indicating cooperativity between the peptides in stabilizing antibody binding. This also implies that the contact face on both CD55 antigen and 105AD7 is generated by the cooperation of several peptides positioned on two domains in each protein. Thus a human monoclonal anti-idiotypic antibody generated by a cancer patient is able to show both amino acid and structural homology with the complement regulatory protein CD55. These findings help identify the mechanism by which a human anti-idiotypic antibody is able to mimic a tumor-associated antigen and stimulate anti-tumor B and T cell responses.

CONTROLLED TERM:
*Adenocarcinoma: IM, immunology
Adenocarcinoma: RI, radionuclide imaging
Adenocarcinoma: SC, secondary
Adenocarcinoma: TH, therapy
Adjuvants, Immunologic: CH, chemistry
Adjuvants, Immunologic: TU, therapeutic use
Amino Acid Sequence
Animals
Antibodies, Anti-Idiotypic: CH, chemistry
Antibodies, Anti-Idiotypic: GE, genetics
*Antibodies, Anti-Idiotypic: TU, therapeutic use
Antibodies, Monoclonal: CH, chemistry
Antibodies, Monoclonal: GE, genetics
*Antibodies, Monoclonal: IM, immunology
Antibodies, Neoplasm: BI, biosynthesis
Antibodies, Neoplasm: DU, diagnostic use
*Antibodies, Neoplasm: IM, immunology
Antigen-Antibody Reactions
Antigens, CD: CH, chemistry
Antigens, CD46
***Antigens, CD55: CH, chemistry**
Antigens, CD55: GE, genetics
Antigens, CD55: IM, immunology
*Antigens, Neoplasm: CH, chemistry
Antigens, Neoplasm: GE, genetics
Antigens, Neoplasm: IM, immunology
Binding Sites, Antibody
CHO Cells
Cloning, Molecular
*Colorectal Neoplasms: IM, immunology
Colorectal Neoplasms: TH, therapy
Cricetinae
Genes, Immunoglobulin
Humans
Immune Sera: IM, immunology
Immunity, Cellular
Immunoglobulin Variable Region: GE, genetics

Liver Neoplasms: RI, radionuclide imaging
Liver Neoplasms: SC, secondary
Membrane Glycoproteins: CH, chemistry
Mice
Mice, Inbred BALB C
Models, Molecular
Molecular Mimicry
Molecular Sequence Data
Peptide Fragments: CH, chemistry
Protein Conformation
Protein Structure, Tertiary
Radioimmunodetection
Recombinant Fusion Proteins: CH, chemistry
Recombinant Fusion Proteins: IM, immunology
Research Support, Non-U.S. Gov't
Sequence Alignment
Sequence Homology, Amino Acid
Transfection

CHEMICAL NAME: 0 (Adjuvants, Immunologic); 0 (Antibodies, Anti-Idiotypic); 0 (Antibodies, Monoclonal); 0 (Antibodies, Neoplasm); 0 (Antigens, CD); 0 (Antigens, CD46); 0 (Antigens, CD55); 0 (Antigens, Neoplasm); 0 (Binding Sites, Antibody); 0 (CD46 protein, human); 0 (Immune Sera); 0 (Immunoglobulin Variable Region); 0 (Mcp protein, mouse); 0 (Membrane Glycoproteins); 0 (Peptide Fragments); 0 (Recombinant Fusion Proteins)

L87 ANSWER 5 OF 28 MEDLINE on STN
ACCESSION NUMBER: 92302264 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 1376921
TITLE: Random PCR mutagenesis screening of secreted proteins by direct expression in mammalian cells.
AUTHOR: Rice G C; Goeddel D V; Cachianes G; Woronicz J; Chen E Y; Williams S R; Leung D W
CORPORATE SOURCE: Department of Cell Biology, Genentech, Inc., South San Francisco, CA 94080.
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1992 Jun 15) Vol. 89, No. 12, pp. 5467-71.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199207
ENTRY DATE: Entered STN: 31 Jul 1992
Last Updated on STN: 6 Feb 1998
Entered Medline: 21 Jul 1992

ABSTRACT:
We have developed a general method for screening randomly mutagenized expression libraries in mammalian cells by using fluorescence-activated cell sorting (FACS). The cDNA sequence of a secreted protein is randomly mutagenized by PCR under conditions of reduced Taq polymerase fidelity. The mutated DNA is inserted into an expression vector encoding the membrane glycophospholipid anchor sequence of decay-accelerating factor (DAF) fused to the C terminus of the secreted protein. This results in expression of the protein on the cell surface in transiently transfected mammalian cells, which can then be screened by FACS. This method was used to isolate mutants in the kringle 1 (K1) domain of tissue plasminogen activator (t-PA) that would no longer be recognized by a specific monoclonal antibody (mAb387) that inhibits

binding of t-PA to its clearance receptor. DNA sequence analysis of the mutants and localization of the mutated residues on a three-dimensional model of the K1 domain identified three key discontinuous amino acid residues that are essential for mAb387 binding. Mutants with changes in any of these three residues were found to have reduced binding to the t-PA receptor on human hepatoma HepG2 cells but to retain full clot lysis activity.

CONTROLLED TERM: Amino Acid Sequence
Animals
Antibodies, Monoclonal
Antigens, CD55
Blood Proteins: GE, genetics
Carcinoma, Hepatocellular
Cell Line
DNA: GE, genetics
Epitopes: AN, analysis
Flow Cytometry
Gene Library
Humans
Liver Neoplasms
Mammals
*Membrane Proteins: GE, genetics
Membrane Proteins: ME, metabolism
Models, Molecular
*Mutagenesis, Site-Directed
*Polymerase Chain Reaction: MT, methods
Protein Conformation
Recombinant Fusion Proteins: ME, metabolism
Restriction Mapping
*Tissue Plasminogen Activator: GE, genetics
Tissue Plasminogen Activator: ME, metabolism
*Transfection
X-Ray Diffraction

CAS REGISTRY NO.: 9007-49-2 (DNA)
CHEMICAL NAME: 0 (Antibodies, Monoclonal); 0 (Antigens, CD55); 0 (Blood Proteins); 0 (Epitopes); 0 (Membrane Proteins); 0 (Recombinant Fusion Proteins); EC 3.4.21.68 (Tissue Plasminogen Activator)

L87 ANSWER 6 OF 28 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2004:432678 CAPLUS Full-text
DOCUMENT NUMBER: 141:86434
TITLE: Decay accelerating factor and colorectal cancer
AUTHOR(S): Gao, Xue-qin; Lu, Yan-qin; Han, Jin-xiang
CORPORATE SOURCE: Shangdong Medical & Biotechnology Center, Shandong Academy of Medical Sciences, Jinan, 250062, Peop. Rep. China
SOURCE: Chinese Journal of Cancer Research (2004), 16(1), 73-77
CODEN: CJCRFH; ISSN: 1000-9604
PUBLISHER: Chinese Journal of Cancer Research
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
ED Entered STN: 28 May 2004
AB A review. To review the significance of decay accelerating factor (DAF) in colorectal cancer, the authors searched the data from PubMed and selected the related articles for review. It was found that DAF were expressed in the adenomas and adenocarcinoma of colorectal tissues. The release of DAF in the

disease. Its detection in the stool of the patients was also detectable. It increased more significantly in the stool of patients with colorectal cancer than other gastrointestinal cancer. Its detection by ELISA method may render a good test for the noninvasive diagnosis of colorectal cancer. It can be concluded that DAF is expressed extensively in colorectal cancer. The detection of DAF released in the stool of colorectal cancer patients may be a good noninvasive method for the diagnosis of colorectal cancer.

CC 14-0 (Mammalian Pathological Biochemistry)

IT Carcinoma
Intestine, neoplasm
(colorectal **adenocarcinoma**; **decay accelerating factor** and colorectal cancer)

REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L87 ANSWER 7 OF 28 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 2

ACCESSION NUMBER: 2001:851500 CAPLUS Full-text
DOCUMENT NUMBER: 135:368932
TITLE: Diagnostic method for screening complement regulatory protein levels
INVENTOR(S): Martens, Mark G.; Kaul, Anil K.; Kaul, Rashmi
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 33 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001088537	A1	20011122	WO 2001-US14769	20010509
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2003129677	A1	20030710	US 2002-292130	20021112
PRIORITY APPLN. INFO.:			US 2000-203967P	P 20000512
			WO 2001-US14769	A1 20010509

ED Entered STN: 23 Nov 2001

AB The invention provides for a method for the early diagnosis of a premalignant lesion, prognosis of a malignant lesion and a kit for use in more rapid identification of predisposition for malignancy. Endometrial tissue samples from patients with benign endometrium (controls) and patients with biopsy-proven adenocarcinoma of the endometrium were analyzed by immunohistochem. staining and image anal. For all four of the complement regulatory protein levels studied (CD35, CD46, CD55, and CD59), there was a statistically significant difference in quant. protein expression between benign and malignant endometrial samples.

IC ICM G01N033-53

CC 9-10 (Biochemical Methods)
Section cross-reference(s): 14

ST early diagnosis complement regulatory protein premalignant lesion; cancer early diagnosis complement regulatory protein; CD35 CD46 **CD55** CD59 endometrium **adenocarcinoma**

IT CD antigens
RL: ANT (Analyte); BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); OCCU (Occurrence); PROC (Process); USES (Uses)
(**CD55**, as complement regulatory protein; diagnostic method for screening complement regulatory protein levels)
IT Uterus, neoplasm
(endometrium, **adenocarcinoma**; diagnostic method for screening complement regulatory protein levels)
IT Tumor necrosis factors
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(modulation of **CD55** in MCF-7 breast cancer cells; diagnostic method for screening complement regulatory protein levels)
IT Mammary gland
(neoplasm, TNF-α modulation of **CD55** in estrogen-primed MCF-7 cells; diagnostic method for screening complement regulatory protein levels)
IT 50-28-2, β-Estradiol, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(TNF-α modulation of **CD55** in MCF-7 breast cancer cells primed with; diagnostic method for screening complement regulatory protein levels)
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L87 ANSWER 8 OF 28 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 4
ACCESSION NUMBER: 2000:628180 CAPLUS Full-text
DOCUMENT NUMBER: 133:221600
TITLE: Antibodies for cancer therapy and diagnosis
INVENTOR(S): Carter, Paul J.; Ridgway, John B.
PATENT ASSIGNEE(S): Genentech, Inc., USA
SOURCE: PCT Int. Appl., 52 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000052054	A2	20000908	WO 2000-US5352	20000229
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2361877	A1	20000908	CA 2000-2361877	20000229
EP 1157041	A2	20011128	EP 2000-912115	20000229
EP 1157041	B1	20050601		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002543044	T	20021217	JP 2000-602278	20000229

AU 770952	B2	20040311	AU 2000-33898	20000229
AT 296839	T	20050615	AT 2000-912115	20000229
PT 1157041	T	20051031	PT 2000-912115	20000229
EP 1591456	A1	20051102	EP 2005-8362	20000229
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
ES 2243240	T3	20051201	ES 2000-912115	20000229
US 2003219434	A1	20031127	US 2003-447331	20030528
PRIORITY APPLN. INFO.:			US 1999-122262P	P 19990301
			EP 2000-912115	A3 20000229
			US 2000-515825	A3 20000229
			WO 2000-US5352	W 20000229

ED Entered STN: 10 Sep 2000

AB The present application describes a method for making antibodies which can be used for cancer diagnosis or therapy. The application also discloses a method for identifying an antigen which is differentially expressed on the surface of two or more distinct cell populations. The application addnl. describes human antibodies directed against decay accelerating factor (DAF), as well as therapeutic compns. comprising such antibodies. Moreover, the application disclosed a method of treating lung cancer with antibodies directed against DAF.

IC ICM C07K016-00

CC 15-3 (Immunochemistry)
Section cross-reference(s): 3

IT Lung, neoplasm
(**adenocarcinoma**; anti-**decay-accelerating factor** antibodies for cancer therapy and diagnosis)

L87 ANSWER 9 OF 28 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 6

ACCESSION NUMBER: 1996:438602 CAPLUS Full-text

DOCUMENT NUMBER: 125:84177

TITLE: Characterization of the complement-regulatory proteins **decay-accelerating factor** (DAF, CD55) and membrane cofactor protein (MCP, CD46) on a human colonic **adenocarcinoma** cell line

AUTHOR(S): Bjoere, Line; Jensen, Tone Skeie; Matre, Roald

CORPORATE SOURCE: Gade Institute, University Bergen, Bergen, N-5021, Norway

SOURCE: Cancer Immunology Immunotherapy (1996), 42(3), 185-192
CODEN: CIIMDN; ISSN: 0340-7004

PUBLISHER: Springer

DOCUMENT TYPE: Journal

LANGUAGE: English

ED Entered STN: 25 Jul 1996

AB To avoid destruction by complement, normal and malignant cells express membrane glycoproteins that restrict complement activity. These include decay-accelerating factor (DAF, CD55), membrane cofactor protein (MCP, CD46), and protectin (CD59), which are all expressed on colonic adenocarcinoma cells in situ. Here, the authors characterized the C3/C5 convertase regulators DAF and MCP on the human colonic adenocarcinoma cell line HT29. DAF is a glycosyl-phosphatidylinositol-anchored 70-kDa glycoprotein. Blocking expts. with F(ab')2 fragments of the anti-DAF monoclonal antibody BRIC 216 showed that DAF modulates the degree of C3 deposition and mediates resistance to complement-mediated killing of the cells. The expression and function of DAF were enhanced by tumor necrosis factor α (TNFα) and interleukin-1β (IL-1β). Cells incubated with interferon γ (IFNγ) did not alter their DAF expression. Two MCP forms were expressed, with mol. masses of approx. 58 kDa and 68 kDa, the lower form predominating. MCP expression was up-regulated by IL-1β, but

not by TNFα or IFNγ. Expression of DAF and MCP promotes resistance of colonic adenocarcinoma cells to complement-mediated damage, and represents a possible mechanism of tumor escape.

CC 15-4 (Immunochemistry)
Section cross-reference(s): 14

ST complement **decay accelerating factor** colon **adenocarcinoma**; membrane cofactor protein MCP colon **adenocarcinoma**

IT **Glycoproteins**, specific or class
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(MCP (membrane cofactor protein), complement **decay-accelerating factor** and membrane cofactor protein expression on human colonic **adenocarcinoma** cells in relation to tumor pathogenesis)

IT Intestine, neoplasm
(colon, **adenocarcinoma**, complement **decay-accelerating factor** and membrane cofactor protein expression on human colonic **adenocarcinoma** cells in relation to tumor pathogenesis)

IT Lymphokines and Cytokines
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(interleukin 1β, complement **decay-accelerating factor** and membrane cofactor protein expression on human colonic **adenocarcinoma** cells response to)

IT Lymphokines and Cytokines
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(tumor necrosis factor-α, complement **decay-accelerating factor** and membrane cofactor protein expression on human colonic **adenocarcinoma** cells response to)

IT Interferons
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(γ, complement **decay-accelerating factor** and membrane cofactor protein expression on human colonic **adenocarcinoma** cells response to)

IT **99085-47-9**, Complement **decay-accelerating factor**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(complement **decay-accelerating factor** and membrane cofactor protein expression on human colonic **adenocarcinoma** cells in relation to tumor pathogenesis)

L87 ANSWER 10 OF 28 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:238155 CAPLUS Full-text
DOCUMENT NUMBER: 144:310062
TITLE: Genes showing altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer
INVENTOR(S): Kloeppel, Guenter; Luettges, Jutta; Kalthoff, Holger; Ammerpohl, Ole; Gruetzmann, Robert; Pilarsky, Christian; Saeger, Hans Detlev; Alldinger, Ingo
PATENT ASSIGNEE(S): Technische Universitaet Dresden, Germany
SOURCE: Ger. Offen., 132 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent

DOCUMENT LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 102004042822	A1	20060316	DE 2004-102004042822	20040831
WO 2006024283	A2	20060309	WO 2005-DE1527	20050826
WO 2006024283	A3	20060831		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: DE 2004-102004042822A 20040831

ED Entered STN: 17 Mar 2006

AB Genes showing altered levels of expression in healthy vs. neoplastic pancreas are identified for use in the diagnosis of cancers including ductal adenocarcinoma; as indicators in screening for effective drugs; and as targets for nucleic acid-based therapies including antisense nucleic acids or siRNA. Gene expression profiling identified 1419 genes showing changes in levels of expression in neoplastic epithelium of which 650 were up-regulated and 769 were down-regulated. Of the 1419 genes, 1267 were not previously known to have any connection with pancreatic neoplasms.

CC 14-1 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 3

IT Cannabinoid receptors
Glycoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(1, gene for, as marker in diagnosis of pancreatic disease; genes showing altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer)

IT **Glycoproteins**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(C4bp (complement C4b-binding protein), β, gene for, as marker in diagnosis of pancreatic disease; genes showing altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer)

IT **Enzymes**, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(DNA helicase **LSH** (lymphoid-specific helicase), gene for, as marker in diagnosis of pancreatic disease; genes showing altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Egr-1, **gene** for, as marker in diagnosis of pancreatic disease; genes showing altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer)

IT **Gene**, animal
RL: ANT (Analyte); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(GPR143, marker in diagnosis of pnacreatic disease; genes showing

altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer)

IT Histones
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(H4, gene for, as marker in diagnosis of pancreatic disease; genes showing altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer)

IT Gene, animal
RL: ANT (Analyte); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(KIF11, marker in diagnosis of pnacreatic disease; genes showing altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer)

IT Glycoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(M6A, gene for, as marker in diagnosis of pancreatic disease; genes showing altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer)

IT Glycoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(M6B, gene for, as marker in diagnosis of pancreatic disease; genes showing altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer)

IT P-glycoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(MDR1, gene for, as marker in diagnosis of pancreatic disease; genes showing altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer)

IT P-glycoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(MDR3, gene for, as marker in diagnosis of pancreatic disease; genes showing altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer)

IT Gene, animal
RL: ANT (Analyte); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(RELN, marker in diagnosis of pnacreatic disease; genes showing altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer)

IT Gene, animal
RL: ANT (Analyte); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(SEPP1, marker in diagnosis of pnacreatic disease; genes showing altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer)

IT 9000-83-3, ATPase 9001-08-5, Butyrylcholinesterase 9001-15-4, Creatine kinase 9001-29-0, Coagulation factor X 9001-50-7, Glyceraldehyde-3-phosphate dehydrogenase 9001-52-9, Fructose-1,6- bisphosphatase 9001-59-6, Pyruvate kinase 9001-60-9, Lactate dehydrogenase 9001-62-1 9001-77-8, Acid phosphatase 9001-80-3, Phosphofructokinase 9001-83-6, Phosphoglycerate kinase 9001-84-7, Phospholipase A2 9001-85-8, Lysophospholipase 9002-06-6, Thymidine kinase 9012-34-4, Acylphosphatase 9013-08-5, Phosphoenolpyruvate carboxykinase 9013-18-7, Long chain acyl-CoA synthetase 9013-55-2, Blood-coagulation factor XI 9013-66-5, GLUTATHIONE PEROXIDASE 9013-81-4, IMP cyclohydrolase 9014-08-8, Enolase 9014-18-0, Nicotinamide nucleotide transhydrogenase 9014-34-0, Stearoyl-CoA desaturase 9014-46-4, Transaldolase 9015-82-1, Angiotensin I converting enzyme 9023-46-5, Threonyl-tRNA synthetase 9023-53-4, Phosphoribosylaminoimidazole synthetase 9023-67-0, Phosphoribosylaminoimidazole succinocarboxamide

synthetase 9023-78-3, Triosephosphate isomerase 9024-52-6, Fructose-bisphosphate aldolase 9024-58-2, Glutamate decarboxylase 9024-61-7, Histidine decarboxylase 9025-77-8, Phosphatidic acid phosphatase 9026-00-0, Bile salt-stimulated lipase) 9026-51-1, Nucleoside-diphosphate kinase 9027-35-4, Glycine amidinotransferase 9027-73-0, 5'-Nucleotidase 9027-80-9, Adenine phosphoribosyltransferase 9028-06-2, Proline 4-hydroxylase 9029-07-6, Aldehyde oxidase 9029-73-6, Phenylalanine hydroxylase 9029-83-8, Serine hydroxymethyltransferase 9029-88-3, Acetylglutamate synthase 9029-95-2, Glycine acyltransferase 9030-22-2, Uridine phosphorylase 9031-37-2, Ferroxidase) 9031-61-2, Thymidylate synthetase 9031-86-1, Aspartoacylase 9032-01-3, Phosphoribosylglycinamide synthetase 9032-02-4, Phosphoribosylglycinamide formyltransferase 9032-03-5, 5-Aminoimidazole-4-carboxamide ribonucleotideformyltransferase 9032-04-6, Phosphoribosylaminoimidazole carboxylase 9032-20-6, NAD(P)H dehydrogenase, quinone 9032-29-5, Dihydrolipoamide acetyltransferase 9032-66-0, NAD kinase 9032-69-3, NAD synthetase 9032-73-9, Monocyte/macrophage serine esterase 1) 9035-39-6, Cytochrome b5 9035-81-8, Antitrypsin 9036-09-3, Chymotrypsin C 9039-53-6, Urokinase 9045-31-2, γ-Butyrobetaine hydroxylase 9047-64-7, Ribonucleotide reductase 9054-63-1, Aminopeptidase N 9055-66-7, Phenylalanine-tRNA synthetase 9059-11-4, Amine oxidase 9074-87-7, γ-Glutamyl hydrolase 9075-15-4, UDP-N-acetyl-α-D-galactosamine: protein N-acetylgalactosaminyltransferase 9075-59-6, Glutaminyl-tRNA synthetase 9075-65-4, Glycerol-3-phosphate dehydrogenase 9081-34-9, Steroid-5α-reductase 12651-27-3, Transcobalamin I 37211-59-9, GDP-mannose 4,6-dehydratase 37228-72-1, Glycine methyltransferase 37237-43-7, β1,4-Galactosyltransferase-I 37255-38-2, Glutaryl-Coenzyme A dehydrogenase 37257-21-9, Glutaminyl-peptide cyclotransferase 37270-64-7, Acyl-CoA hydrolase 37278-25-4, Ribonuclease t2 37288-40-7 39419-81-3, Holocarboxylase synthetase 50864-48-7, Sphingosine kinase 1 52410-46-5, Sterol Δ8→7-isomerase 55126-92-6, Colipase 56645-49-9, Cathepsin G 58319-92-9, ADP-ribosyltransferase 59536-74-2, Long chain acyl-Coenzyme A dehydrogenase 62229-50-9, Epidermal growth factor 65802-85-9, Prostaglandin D2 synthase 65979-40-0 68651-94-5 72162-84-6, Prolyl endopeptidase 72162-89-1, TRNA-guanine transglycosylase) 74812-49-0, E3 Ubiquitin protein ligase 75536-80-0, Peptidyl arginine deiminase 76774-39-5, Ribonuclease L 80295-56-3, Complement C6 80295-57-4, Complement C7 80295-65-4, Complement factor H 81181-75-1, Lewis type 1 antigen synthase 82599-72-2, Polynucleotide kinase 3'-phosphatase 83589-04-2, Chondroitin sulfotransferase 87397-91-9, Thymosin β10 91386-47-9, Mesotrypsin 95076-93-0, Peptidyl prolyl isomerase 97089-82-2, 6-Pyruvoyl-tetrahydropterin synthase **99085-47-9**, Complement **decay-accelerating factor** 99194-04-4, Cystatin B 102484-71-9, Cystatin SN 102576-81-8, Acetylglucosaminyltransferase I 104645-76-3, Phosphatidylinositol-4-phosphate 5-kinase 106602-62-4, Islet amyloid polypeptide 107544-29-6, Cystatin A 109136-49-4, Ubiquitin specific protease 110071-61-9, Myristoylating enzymes 110910-42-4, Cathepsin E 111693-80-2, Inositol polyphosphate-4-phosphatase 111745-44-9, Neuromedin U 114051-78-4, Lck kinase 117628-82-7, Follistatin 117698-12-1, Paraoxonase 120038-28-0, Carboxypeptidase M 125692-40-2, Endothelin 3 142805-56-9, DNA topoisomerase II 143375-33-1, Neurotrophin 5 145267-01-2, Stromelysin 3 148047-29-4, Gene TEK protein tyrosine kinase 149371-24-4, Neurolysin 151662-24-7, Paired basic amino acid cleaving enzyme 4 151662-33-8, Pregnancy-associated plasma protein A 152166-53-5, Neurotrophic factor receptor tyrosine kinase 152787-71-8, TTK kinase 153190-51-3, Protein tyrosine kinase PTK6 155807-64-0, Flap

structure-specific endonuclease 1 158254-85-4, Lysophosphatidic acid phosphatase 160477-63-4, Tissue factor pathway inhibitor 2 160477-87-2, PITSLRE protein kinase 165245-94-3, NIMA-related kinase 2 167397-96-8, Interleukin-1 receptor-associated kinase 1 169592-65-8, β-Carotene 4,4'-dioxygenase 172306-54-6, LIM kinase 2 178037-70-2, Protein kinase SGK 178303-46-3, BMX tyrosine kinase 181186-98-1, Carboxypeptidase A2 182372-11-8, Meltrin α 186270-49-5, Angiopoietin 1 188364-82-1, Neuroserpin 190606-22-5, Maternal embryonic leucine zipper kinase 192588-76-4, CASP8 and FADD-like apoptosis regulator 202420-40-4, Serine/threonine protein kinase 11 204655-80-1, Serine proteinase 25 206566-35-0, Molybdenum cofactor sulfurase 241475-68-3, ADAMTS-1 246521-08-4, Hyaluronan binding protein 2 251104-39-9, Tolloid-like 2 252351-00-1, Metalloproteinase ADAM8 288265-32-7, ADAM28 288307-53-9, Inositol 1,3,4-trisphosphate 5/6 kinase 306298-47-5, Dual specificity phosphatase 1 330197-29-0, Cyclin-dependent kinase 7 330207-13-1, Cytochrome P 450 2C8 334993-12-3, Kallikrein 10 342900-25-8, Kallikrein 12 354807-39-9 361186-67-6, MAPK phosphatase 7 361540-77-4, Protein phosphatase 3 362674-81-5, Protein phosphatase 2A 364367-46-4, Protein phosphatase 4 370088-29-2, Mitogen-activated protein kinase kinase kinase kinase 4 371761-91-0, (Survivin) 376596-92-8, β-Defensin 1 388138-21-4, KiSS-1 metastasis-suppressor 389069-73-2, Kallikrein 1 404843-77-2, Reelin 415715-09-2, BMP2 inducible kinase 458560-40-2, Serine/threonine protein kinase 6 473573-11-4, Protein kinase Rio2 475678-93-4, Short chain dehydrogenase reductase 489461-60-1, Trypsin-2 495418-42-3, Cytochrome P 450 4X1 690230-94-5, Metalloproteinase ADAM33
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(gene for, as marker in diagnosis of pancreatic disease; genes showing altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer)

IT 72162-83-5, **Glycoprotein** sulfotransferase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**glycoprotein** sulfotransferase, gene for, as marker in diagnosis of pancreatic disease; genes showing altered levels of expression in pancreatic disease and their use in diagnosis and prognosis of pancreatic cancer)

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L87 ANSWER 11 OF 28 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:1167571 CAPLUS Full-text
DOCUMENT NUMBER: 146:59916
TITLE: Differential expression of insulin-like growth factor binding protein-5 in pancreatic adenocarcinomas: identification using DNA microarray
AUTHOR(S): Johnson, Sarah K.; Dennis, Richard A.; Barone, Gary W.; Lamps, Laura W.; Haun, Randy S.
CORPORATE SOURCE: Department of Pathology, University of Arkansas for Medical Sciences, Little Rock, AR, USA
SOURCE: Molecular Carcinogenesis (2006), 45(11), 814-827
CODEN: MOCAE8; ISSN: 0899-1987
PUBLISHER: Wiley-Liss, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 07 Nov 2006
AB Pancreatic ductal adenocarcinoma (PDAC) is characterized by its aggressiveness and resistance to both radiation and chemotherapeutic treatment. To better understand the mol. pathogenesis of pancreatic cancer, DNA array technol. was employed to identify genes differentially expressed in pancreatic tumors when

compared to non-malignant pancreatic tissues. RNA isolated from 11 PDACs and 14 non-malignant bulk pancreatic duct specimens was used to probe Affymetrix U95A DNA arrays. Genes that displayed at least a fourfold differential expression were identified and real-time quant. PCR was used to verify the differential expression of selected upregulated genes. Interrogation of the DNA array revealed that 73 genes were upregulated in PDACs and 77 genes were downregulated. The majority of the 150 genes identified have not been previously reported to be differentially expressed in pancreatic tumors, although a number of the upregulated transcripts have been reported previously. Immunohistochem. was used to correlate calponin and insulin-like growth factor binding protein-5 (IGFBP-5) RNA levels with protein expression in PDACs and revealed peritumoral calponin staining in the reactive stroma and intense focal staining of islets cells expressing IGFBP-5 at the edge of tumors; thus implicating the interplay of various cell types to promote neoplastic cell growth within pancreatic carcinomas. As a potential modulator of cell proliferation, the overexpression of IGFBP-5 may, therefore, play a significant role in the malignant transformation of normal pancreatic epithelial cells.

CC 14-1 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 3

IT **99085-47-9, Decay accelerating factor**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(differential expression of insulin-like growth factor binding protein-5 and other genes in pancreatic **adenocarcinomas**)

REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L87 ANSWER 12 OF 28 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:324225 CAPLUS Full-text
DOCUMENT NUMBER: 141:86940
TITLE: Enhanced expression of decay-accelerating factor, a complement-regulatory protein, in the specialized intestinal metaplasia of Barrett's esophagus
AUTHOR(S): Hiraoka, Sakiko; Mizuno, Motowo; Nasu, Junichirou; Okazaki, Hiroaki; Makidono, Chiho; Okada, Hiroyuki; Terada, Ryo; Yamamoto, Kazuhide; Fujita, Teizo; Shiratori, Yasushi
CORPORATE SOURCE: Department of Medicine and Medical Science (Medicine 1), Okayama University Graduate School of Medicine and Dentistry, Okayama, Japan
SOURCE: Journal of Laboratory and Clinical Medicine (2004), 143(4), 201-206
CODEN: JLCMAK; ISSN: 0022-2143
PUBLISHER: Elsevier Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 21 Apr 2004

AB Intestinal-type epithelium in Barrett's esophagus, so-called specialized intestinal metaplasia (SIM), is a risk factor for the development of esophageal adenocarcinoma. Surface expression of decay-accelerating factor (DAF), a complement-regulatory protein, is markedly enhanced in intestinal metaplasia of the gastric mucosa. The authors therefore examined DAF expression in areas of SIM in Barrett's esophagus to determine whether DAF is a biomarker of SIM. The authors obtained 53 endoscopic biopsy specimens from the esophageal columnar mucosae of 45 patients. The authors immunohistochem. examined the distribution of DAF and 2 other complement-regulatory proteins: homologous restriction factor-20 and membrane cofactor protein. The authors also examined the expression of DAF mRNA in SIM with the use of laser-capture microdissection and reverse transcription-polymerase chain reaction. Of the 53 specimens, 10 were found histol. to involve areas of SIM, 41 were SIM-neg.

epithelium, and 2 comprised areas of SIM and SIM-neg. epithelium. DAF staining was negligible in 35 of 43 specimens of the SIM-neg. columnar epithelium, but DAF was strongly stained on the apical surface in all 12 SIM-pos. specimens. In the 2 biopsy specimens in which both SIM and SIM-neg. columnar epithelium were present, DAF staining was confined to the area of SIM. The expression of DAF mRNA was detected significantly more often in SIM than in SIM-neg. columnar epithelium. The authors conclude that DAF may be a surface marker for SIM and therefore useful in the identification of areas of the mucosa at risk for the development of adenocarcinoma in Barrett's esophagus.

CC 14-7 (Mammalian Pathological Biochemistry)

IT Esophagus, disease
(Barrett's syndrome; enhanced expression of **decay-accelerating factor** in specialized intestinal metaplasia of Barrett's esophagus in relation to esophageal **adenocarcinoma** development)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(MCP (membrane cofactor protein); enhanced expression of **decay-accelerating factor** and other complement-regulatory proteins in specialized intestinal metaplasia of Barrett's esophagus in relation to esophageal **adenocarcinoma** development)

IT Esophagus, neoplasm
(**adenocarcinoma**; enhanced expression of **decay-accelerating factor** in specialized intestinal metaplasia of Barrett's esophagus in relation to esophageal **adenocarcinoma** development)

IT Cell membrane
(apical; enhanced expression of **decay-accelerating factor** in specialized intestinal metaplasia of Barrett's esophagus in relation to esophageal **adenocarcinoma** development)

IT CD59 (antigen)
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(enhanced expression of **decay-accelerating factor** and other complement-regulatory proteins in specialized intestinal metaplasia of Barrett's esophagus in relation to esophageal **adenocarcinoma** development)

IT Human
Prognosis
(enhanced expression of **decay-accelerating factor** in specialized intestinal metaplasia of Barrett's esophagus in relation to esophageal **adenocarcinoma** development)

IT Esophagus
(epithelium, columnar; enhanced expression of **decay-accelerating factor** in specialized intestinal metaplasia of Barrett's esophagus in relation to esophageal **adenocarcinoma** development)

IT Carcinoma
(esophageal **adenocarcinoma**; enhanced expression of **decay-accelerating factor** in specialized intestinal metaplasia of Barrett's esophagus in relation to esophageal **adenocarcinoma** development)

IT Epithelium
(esophageal, columnar; enhanced expression of **decay-accelerating factor** in specialized intestinal metaplasia of Barrett's esophagus in relation to esophageal **adenocarcinoma** development)

IT Intestine, disease
(metaplasia; enhanced expression of decay-accelerating factor in specialized intestinal metaplasia of Barrett's esophagus in relation to esophageal adenocarcinoma development)
IT 99085-47-9, Decay accelerating factor
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); BIOL (Biological study); USES (Uses)
(enhanced expression of decay-accelerating factor in specialized intestinal metaplasia of Barrett's esophagus in relation to esophageal adenocarcinoma development)
REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L87 ANSWER 13 OF 28 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:446876 CAPLUS Full-text
DOCUMENT NUMBER: 138:70725
TITLE: Characterization of gene expression profiles in intraductal papillary-mucinous tumors of the pancreas
AUTHOR(S): Terris, Benoit; Blaveri, Ekaterina; Crnogorac-Jurcevic, Tatjana; Jones, Melanie; Missiaglia, Edoardo; Ruszniewski, Philippe; Sauvanet, Alain; Lemoine, Nicholas R.
CORPORATE SOURCE: Cancer Research UK Molecular Oncology Unit, Imperial College School of Medicine at Hammersmith Campus, London, UK
SOURCE: American Journal of Pathology (2002), 160(5), 1745-1754
CODEN: AJPAA4; ISSN: 0002-9440
PUBLISHER: American Society for Investigative Pathology
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 14 Jun 2002
AB The mol. pathol. of precursor lesions leading to invasive pancreatic ductal adenocarcinomas remains relatively unknown. The authors have applied cDNA microarray anal. to characterize gene expression profiles in a series of intraductal papillary-mucinous tumors (IPMTs) of the pancreas, which represents one of the alternative routes of intraepithelial progression to full malignancy in the pancreatic duct system. Using a cDNA microarray containing 4992 human genes, the authors screened a total of 13 IPMTs including nine noninvasive and four invasive cases. Expression change in more than half of the tumors was observed for 120 genes, i.e., 62 up-regulated and 58 down-regulated genes. Some of the up-regulated genes in this study have been previously described in classical pancreatic carcinomas such as lipocalin 2, galectin 3, claudin 4, and cathepsin E. The most highly up-regulated genes in IPMTs corresponded to three members of the trefoil factor family (TFF1, TFF2, and TFF3). Immunohistochem. performed on five genes found to be differentially expressed at the RNA level (TFF1, TFF2, TFF3, lipocalin 2, and galectin 3) showed a good concordance between transcript level and protein abundance, except for TFF2. Hierarchical clustering organized the cases according to the dysplastic and invasive phenotype of the IPMTs. This anal. has permitted us to implicate several genes (caveolin 1, glypican 1, growth arrest-specific 6 protein, cysteine-rich angiogenic inducer 61) in tumor progression. The observation that several genes are differentially expressed both in IPMTs and pancreatic carcinomas suggests that they may be involved at an early stage of pancreatic carcinogenesis.
CC 14-1 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 3
IT Pancreas, neoplasm

(duct cell **adenocarcinoma**; characterization of gene expression profiles in intraductal papillary-mucinous tumors of pancreas)

IT Carcinoma
(pancreatic ductal **adenocarcinoma**; characterization of gene expression profiles in intraductal papillary-mucinous tumors of pancreas)

IT 99085-47-9, **CD55** antigen 110910-42-4, Cathepsin E
147014-97-9, CDK4 kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(characterization of gene expression profiles in intraductal papillary-mucinous tumors of pancreas)

REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L87 ANSWER 14 OF 28 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2001:258387 CAPLUS Full-text
DOCUMENT NUMBER: 135:224861
TITLE: Polymorphic expression of decay-accelerating factor in human colorectal cancer
AUTHOR(S): Nakagawa, Masahiro; Mizuno, Motowo; Kawada, Mikihiro; Uesu, Tokurou; Nasu, Junichirou; Takeuchi, Kazuaki; Okada, Hiroyuki; Endo, Yuichi; Fujita, Teizo; Tsuji, Takao
CORPORATE SOURCE: First Department of Internal Medicine, Okayama University Medical School, Okayama, 700-8558, Japan
SOURCE: Journal of Gastroenterology and Hepatology (2001), 16(2), 184-189
CODEN: JGHEEO; ISSN: 0815-9319
PUBLISHER: Blackwell Science Asia Pty Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 12 Apr 2001
AB We have previously shown that expression of decay-accelerating factor (DAF), a complement regulatory protein, is enhanced immunohistochem. on the luminal surface of cancer glands in human colorectal cancer and is detected in stool specimens of patients with colorectal cancer. The amount of DAF present in the stools might be influenced by the stability of DAF on the cell surface which is regulated by biochem. properties such as glycosylation of the protein. In the present study, to help elucidate the mechanism for the release of DAF from human colorectal cancers, we biochem. analyzed DAF expression by western and northern blotting by using surgically resected specimens of colorectal cancers. Surgically resected colorectal cancer tissues were obtained from 10 patients. Expression of DAF was determined by western and northern blotting, and glycosylation of DAF protein was analyzed with glycosidase digestion. Northern blot anal. demonstrated that the expression of DAF mRNA in colorectal cancer was enhanced two- to threefold compared with normal tissues. In western blotting, expression of DAF protein in the cancer tissue was increased, and heterogeneity in the apparent mol. weight of DAF was observed among patients. When O-linked sugars were removed, this heterogeneity of DAF size diminished. The polymorphic expression of DAF in colorectal cancer is likely to reflect variability in the O-glycosylation of the protein. We speculate that this variability could affect the stability of DAF on the surfaces of cancer cells and, in turn, the amount of DAF shed into the stools of colorectal cancer patients.
CC 14-1 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 15
IT Intestine, neoplasm
(colorectal **adenocarcinoma**; polymorphic expression of **decay-accelerating factor** in human

colorectal cancer)

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L87 ANSWER 15 OF 28 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1999:344861 CAPLUS Full-text
DOCUMENT NUMBER: 131:4240
TITLE: Immunoglobulin molecules having a synthetic variable region and modified specificity
INVENTOR(S): Burch, Ronald M.
PATENT ASSIGNEE(S): Euro-Celtique, S.A., Bermuda
SOURCE: PCT Int. Appl., 123 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9925378	A1	19990527	WO 1998-US24302	19981113
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2309990	A1	19990527	CA 1998-2309990	19981113
CA 2310269	A1	19990527	CA 1998-2310269	19981113
WO 9925379	A1	19990527	WO 1998-US24303	19981113
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW				
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AU 9914597	A	19990607	AU 1999-14597	19981113
AU 763029	B2	20030710		
AU 9914598	A	19990607	AU 1999-14598	19981113
AU 737457	B2	20010823		
EP 1030684	A1	20000830	EP 1998-958584	19981113
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
EP 1032420	A1	20000906	EP 1998-958583	19981113
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001526021	T	20011218	JP 2000-520811	19981113
BR 9815289	A	20011226	BR 1998-15289	19981113
BR 9815580	A	20020129	BR 1998-15580	19981113
JP 2002507544	T	20020312	JP 2000-520812	19981113
ZA 9900048	A	19990708	ZA 1999-48	19990105
ZA 9900049	A	20000309	ZA 1999-49	19990105
IN 1999MA00038	A	20050304	IN 1999-MA38	19990107
US 2002028469	A1	20020307	US 2001-963232	20010926
CN 1561287	A	20050105	CN 2002-819009	20020828
AU 2003252902	A1	20031106	AU 2003-252902	20031010

PRIORITY APPLN. INFO.: US 1997-65716P P 19971114
US 1998-81403P P 19980410
US 1998-191780 A1 19981113
WO 1998-US24302 W 19981113
WO 1998-US24303 W 19981113
US 2001-963232 A 20010926

ED Entered STN: 07 Jun 1999

AB The invention provides modified Ig mols., particularly antibodies, that immunospecifically bind a first member of a binding pair which binding pair consists of the first member and a second member, which Igs have a variable domain containing one or more complimentary determining regions that contain the amino acid sequence of a binding site for the second member of the binding pair. The first member is a tumor antigen or an antigen of an infectious disease agent, and the second member is a mol. on the surface of an immune cell. The invention further provides for therapeutic and diagnostic use of the modified Ig.

IC ICM A61K039-395
ICS C12N005-12; C12N015-13; C07K016-42; C07K016-08; C07K016-30

CC 15-3 (Immunochemistry)
Section cross-reference(s): 3

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(195 transmissible gastroenteritis; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(38 swine rotavirus; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(48 and 53; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(55, bovine viral diarrhea virus; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(B, equine herpes virus type I; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(E, pseudorabies virus; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT Human respiratory syncytial virus
(G **glycoprotein**; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(G, human respiratory syncytial virus; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(HN (hemagglutinin-neuraminidase); modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(LPS-LBP (lipopolysaccharide-containing lipopolysaccharide-binding protein), receptors, antigen CD14-containing; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, general, biological studies
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(La Crosse virus; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(MCP (membrane cofactor protein), receptor; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(MCP (membrane cofactor protein); modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT Carcinoma
(**adenocarcinoma**, antigen; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT Human immunodeficiency virus 1
(envelope **glycoproteins**; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(gB herpes simplex virus type 2; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(gD, equine herpes virus type I; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(gH, pseudorabies virus; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(gI, infectious laryngotracheitis virus; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT Porcine rotavirus
(**glycoprotein** 38; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT Bovine diarrhea virus
(**glycoprotein** 55; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT Equid herpesvirus 1
(**glycoprotein** B; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT Bovine herpesvirus 1
(**glycoprotein** E; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT Gallid herpesvirus 1
(**glycoprotein** G; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT Human herpesvirus 2
(**glycoprotein** gB; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT La Crosse virus
(**glycoprotein**; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(gp39; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(gp46, surface; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(gp70; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT **Glycoproteins**, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(neoglycoproteins; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious

agent and surface mol. of immune cell)

IT Bovine diarrhea virus
(neonatal glycoprotein; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

IT 9026-43-1, Protein kinase 55717-54-9, N-Acetyl-9-O-acetylneuraminic acid 99085-47-9, Decay-accelerating factor
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(receptor; modified Ig mols. having a synthetic variable region and modified specificity for tumor antigen or antigen of infectious agent and surface mol. of immune cell)

REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L87 ANSWER 16 OF 28 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1998:703347 CAPLUS Full-text
DOCUMENT NUMBER: 130:108960
TITLE: The complement regulatory proteins CD46 and CD59, but not CD55, are highly expressed by glandular epithelium of human breast and colorectal tumor tissues
AUTHOR(S): Thorsteinsson, L.; O'Dowd, G. M.; Harrington, P. M.; Johnson, P. M.
CORPORATE SOURCE: Cancer Tissue Bank Research Centre, and Departments of Immunology and, University of Liverpool, Liverpool, UK
SOURCE: APMIS (1998), 106(9), 869-878
CODEN: APMSEL; ISSN: 0903-4641
PUBLISHER: Munksgaard International Publishers Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 05 Nov 1998
AB Three of the proteins protecting cells from autologous lysis by complement are: membrane cofactor protein (MCP; CD46), an inhibitor of the membrane attack complex formation (CD59), and decay accelerating factor (DAF; CD55). We have investigated the expression of these proteins in breast and colorectal carcinoma by immunohistochem. and immunoblotting of breast tissue for CD46. CD46 was consistently and strongly expressed in the epithelial compartment in 26/28 ductal carcinomas of the breast, 9/9 fibroadenomas, and 9/11 cases of control non-neoplastic breast tissue. CD59 showed a similar degree of expression in the fibroadenomas (9/9), but was less strongly expressed in carcinomatous (22/28) and control (5/11) tissues. In marked contrast, no CD55 expression was detected in tissue from 15 ductal carcinomas. Immunoblotting of breast tissue for CD46 showed the same size of the mol. as for lymphocytes. It had however considerably stronger expression in tumor tissue than in non-neoplastic tissue. CD46 and CD59 were either lacking or only weakly expressed in the epithelial component of control colorectal mucosa: 2/15 and 5/15, resp. In contrast, tissue samples from colorectal adenocarcinomas showed clear staining for both CD59 (10/18) and, more markedly, CD46 (15/18). There was no association between the pattern or intensity of CD46 and CD59 expression and tumor differentiation. As the complement regulatory proteins CD46 and CD59 are also strongly expressed by trophoblast at the feto-maternal tissue interface, these results support the concept that similar mechanisms are employed both by the genetically dissimilar fetus and certain tumors to evade immune attack by their host.
CC 15-4 (Immunochemistry)
ST CD46 CD59 breast carcinoma immunosuppression; colorectal adenocarcinoma CD46 CD59 immunosuppression
IT Glycoproteins, specific or class
RL: BOC (Biological occurrence); BSU (Biological study, unclassified);

BIOL (Biological study); OCCU (Occurrence)
(MCP (membrane cofactor protein); complement regulatory proteins CD46 and CD59, but not CD55, are highly expressed by glandular epithelium of human breast and colorectal tumor tissues)

IT Intestine, neoplasm
(colorectal **adenocarcinoma**; complement regulatory proteins CD46 and CD59, but not **CD55**, are highly expressed by glandular epithelium of human breast and colorectal tumor tissues)

IT Immunosuppression
(complement regulatory proteins CD46 and CD59, but not **CD55**, are highly expressed by glandular epithelium of human breast and colorectal tumor tissues)

IT CD59 (antigen)
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(complement regulatory proteins CD46 and CD59, but not **CD55**, are highly expressed by glandular epithelium of human breast and colorectal tumor tissues)

IT Mammary gland
(ductal carcinoma; complement regulatory proteins CD46 and CD59, but not **CD55**, are highly expressed by glandular epithelium of human breast and colorectal tumor tissues)

IT Mammary gland
(fibroadenoma; complement regulatory proteins CD46 and CD59, but not **CD55**, are highly expressed by glandular epithelium of human breast and colorectal tumor tissues)

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L87 ANSWER 17 OF 28 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1996:160210 CAPLUS Full-text
DOCUMENT NUMBER: 124:258031
TITLE: Differential expression of complement proteins and regulatory **decay accelerating factor** in relation to differentiation of cultured human colon **adenocarcinoma** cell lines
AUTHOR(S): Bernet-Camard, M.-F.; Coconnier, M.-H.; Hudault, S.; Servin, A. L.
CORPORATE SOURCE: UPS de Pharmacie, Paris-XI, Chatenay-Malabry, F-92296, Fr.
SOURCE: Gut (1996), 38(2), 248-53
CODEN: GUTTAK; ISSN: 0017-5749
PUBLISHER: BMJ Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 19 Mar 1996
AB Self protection of host cells against inadvertent injury resulting from attack by autologous complement proteins is well reported for vascular epithelium. In intestinal epithelium, the expression of C complement proteins and regulatory proteins remains currently poorly reported. This study looked at the distribution of C complement proteins and regulatory decay accelerating factor (DAF) in four cultured human intestinal cell lines of embryogenic or colon cancer origins. C3 and C4 proteins and DAF were widely present in human colon adenocarcinoma T84, HT-29 glc-/+ cells compared with human embryonic INT407 cells. In contrast, no expression of C5, C5b-9, and CR1 was seen for any of the cell lines. Taking advantage of the Caco-2 cells, which spontaneously differentiate in culture, it was seen that the C3, C4, and DAF were present in undifferentiated cells and that their expression increased as a function of the cell differentiation. These results, taken together with

other reports on the presence of C complement proteins and DAF in the intestinal cells infer that the expression of regulatory C complement proteins develops in parallel with the expression of C proteins to protect these cells against the potential injury resulting from the activation of these local C proteins. Moreover, the finding that the pathogenic C1845 Escherichia coli binds to the membrane bound DAF in the cultured human intestinal cells synthesizing locally C proteins and regulatory C proteins supports the hypothesis that E. coli could promote inflammatory disorders by blocking local regulatory protein function.

CC 15-4 (Immunochemistry)
Section cross-reference(s): 10

L87 ANSWER 18 OF 28 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1993:667694 CAPLUS Full-text
DOCUMENT NUMBER: 119:267694
TITLE: Levels of complement regulatory molecules in lung cancer: disappearance of the D17 epitope of CD55 in small-cell carcinoma
AUTHOR(S): Sakuma, Takahiko; Kodama, Ken; Hara, Tomoko; Eshita, Yoshimi; Shibata, Nobuhiko; Matsumoto, Misako; Seya, Tsukasa; Mori, Yoichi
CORPORATE SOURCE: Dep. Intern. Med., Cent. Adult Dis. Osaka, Osaka, 537, Japan
SOURCE: Japanese Journal of Cancer Research (1993), 84(7), 753-9
CODEN: JJCREP; ISSN: 0910-5050
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 25 Dec 1993
AB The levels of complement-regulatory mols. (complement receptor type one [CRI], decay-accelerating factor [DAF], membrane cofactor protein [mcp], and an inhibitor of membrane attack complex [CD59]) in lung cancer cells were analyzed to investigate the relation between their expression and histol. subtypes, and the possibility of homologous complement deposition on cancer cells. In 25 cell lines (10 adenocarcinoma, 3 large-cell carcinoma, 7 small-cell lung cancer [SCLC], and 5 squamous cell carcinoma), flow cytometric anal. revealed that MCP was expressed in all cell lines, whereas none of the cell lines was CR1-pos., CD59 was detected in all cells. The DAF epitope defined by IA10 was expressed in all cells except one large cell carcinoma cell line. However, another epitope for anti-DAF monoclonal antibody, D17, was not detected in 5 (71.4%) SCLC and in 4 (22.2%) non-small-cell lung cancer. This disparity was seen in most cell lines, irresp. of histol. subtypes. The loss of D17 reactivity seemed to be pertinent to malignant phenotype, because most of the normal pulmonary cells possessed the D17 epitope. Furthermore, a cell line lacking DAF (IA10-/D17-) allowed alternative pathway-mediated homologous complement (C3) deposition after pretreatment with anti-MCP antibody. This raises a new possibility for immuno-targeting of cancer. These cell lines should be useful in studying the biol. of lung cancer.

CC 14-1 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 15
IT **Glycoproteins**, specific or class
RL: BIOL (Biological study)
(MCP (membrane cofactor protein), of lung carcinoma cells, of humans)
IT Lung, neoplasm
(**adenocarcinoma**, complement regulatory proteins of cells of, of humans)
IT Lung, neoplasm
(small-cell carcinoma, complement regulatory proteins and lack of D17 epitope-defined **decay-accelerating factor** of cells of, of humans)

JT 99085 47-9, Decay-accelerating factor
RL: BIOL (Biological study)
(of lung carcinoma cells, of humans, decay-accelerating factor D17 epitope absence in small-cell carcinoma of lung in relation to)

L87 ANSWER 19 OF 28 WPIX COPYRIGHT 2007 THE THOMSON CORP on STN
ACCESSION NUMBER: 2005-435500 [44] WPIX
DOC. NO. CPI: C2005-133663 [44]
DOC. NO. NON-CPI: N2005-353395 [44]
TITLE: System, useful for transdermal delivery of an active agent, comprises an apparatus (capable of generating at least one micro-channel in an area on the skin) and a patch comprising at least one drug reservoir layer
DERWENT CLASS: A18; A23; A25; A96; B04; B07; P34
INVENTOR: LEVIN G; SACKS H
PATENT ASSIGNEE: (TRAN-N) TRANSPHARMA MEDICAL LTD
COUNTRY COUNT: 107

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
WO 2005056075	A2	20050623	(200544)*	EN	53[12]	
EP 1691823	A2	20060823	(200655)	EN		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2005056075	A2	WO 2004-IL1119	20041209
EP 1691823	A2	EP 2004-801605	20041209
EP 1691823	A2	WO 2004-IL1119	20041209

FILING DETAILS:

PATENT NO	KIND		PATENT NO	
EP 1691823	A2	Based on	WO 2005056075	A

PRIORITY APPLN. INFO: IL 2003-159273 20031209
INT. PATENT CLASSIF.:
IPC ORIGINAL: A61K0038-02 [I,A]; A61K0038-28 [I,A]; A61K0038-39 [I,A]; A61N0001-00 [I,A]; A61N0001-30 [I,A]; C07K0014-435 [I,C]; C07K0014-62 [I,A]; C07K0014-78 [I,A]; C07K0002-00 [I,A]
IPC RECLASSIF.: A61K [I,S]; A61M [I,S]
BASIC ABSTRACT:
WO 2005056075 A2 UPAB: 20051222
NOVELTY - System (A) for facilitating transdermal delivery of an active agent (B) through skin comprises an apparatus (I) capable of generating at least one micro-channel in an area on the skin and a patch (II) comprising at least one drug reservoir layer (1), which comprises a polymeric matrix and a pharmaceutical composition comprising an active agent (at least one therapeutic or immunogenic peptide, polypeptide or protein).
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for: (1) (II) adapted for transdermal delivery of (B), comprising (1), which comprises a hydrophilic polymeric matrix and (B) such as a therapeutic or immunogenic

peptide, polypeptide and protein; and (2) a method for sustained transdermal delivery of a therapeutic or immunogenic agent, comprising generating at least one micro-channel in a region on the skin, affixing (II) to the region of skin in which the at least one microchannel is present and achieving a therapeutic blood concentration of the peptide, polypeptide or protein for at least 6 hours.

USE - (A) is useful for sustained transdermal delivery of an active agent such as therapeutic or immunogenic peptide, polypeptide or protein (claimed).

ADVANTAGE - (A) is highly efficient and provides sustained and slow delivery of hydrophilic high **molecular weight** proteins. (I) present in (A) enhances the transdermal delivery of an active agent and (II) present in (A) maintains the stability and activity of the active agent throughout the transdermal delivery, thus maintaining therapeutic blood concentrations for significantly extended periods of time and achieving extended therapeutic effect as compared to that obtained by subcutaneous injection. MANUAL CODE: CPI: A12-V01; B04-B04C; B04-C01; B04-C02A2; B04-C02E3;
B04-C03; B04-G01; B04-H06; B04-J01; B04-K01; B04-L03; B04-L05; B04-N02; B04-N04; **B04-N06**; B12-M02F; B12-M07; B12-M10A; B14-D07C

TECH

INSTRUMENTATION AND TESTING - Preferred Components: (I) comprises an electrode cartridge comprising a plurality of electrodes; and a main unit comprising a control unit, which is adapted to apply electrical energy to the electrodes when the electrodes are in vicinity of the skin, enabling ablation of stratum corneum in an area beneath the electrodes to generate at least one micro-channel. The electrode cartridge is adapted to generate a plurality of micro-channels of uniform shape and dimensions. The electrical energy is of radio frequency. T(1) is formulated in a dry form, semi-dry form, hydrogel and a solution. (B) is growth factors, hormones, cytokines, water-soluble drugs, antigens, antibodies, fragments and their analogs such as insulin, growth hormone (both preferred), proinsulin, follicle stimulating hormone, insulin like growth factor-1, insulin like growth factor-2, platelet derived growth factor, epidermal growth factor, fibroblast growth factors, nerve growth factor, transforming growth factors, tumor necrosis factor, calcitonin, parathyroid hormone, bone morphogenic protein, erythropoietin, hemopoietic growth factors, luteinizing hormone, glucagon, clotting factors, anticlotting factors, atrial natriuretic factor, lung surfactant, plasminogen activators, bombesin, thrombin, enkephalinase, relaxin A-chain, relaxin B-chain, prorelaxin, inhibin, activin, vascular endothelial growth factor, hormone receptors, growth factor receptors, integrins, protein A, protein D, rheumatoid factors, neurotrophic factors, CD proteins, osteoinductive factors, immunotoxins, interferons, colony stimulating factors, interleukins (ILs), superoxide dismutase, surface membrane proteins, T-cell receptors, **decay accelerating factor**, viral antigens, transport proteins, homing receptors, addressins, regulatory proteins, analogs, derivatives and their fragments. (II) further comprises at least one of a backing layer, an adhesive and a rate-controlling layer. The pharmaceutical composition further comprises at least one component such as protease inhibitors, stabilizers, anti-oxidants, buffering agents and preservatives.

POLYMERS - Preferred Composition: he polymeric matrix is hydrophilic biopolymers, hydrophilic synthetic polymers and/or derivatives. The biopolymer is collagens, carrageenans (both preferred), hydroxypropyl cellulose, carboxymethyl cellulose, hydroxyethyl cellulose, chitin, chitosan, alginates, gelatin, pectin, glycosaminoglycans (GAGs), proteoglycans, fibronectins or laminins. The hydrophilic synthetic polymer is polyethylene oxide (preferred), polyglycolic acid (PGA), polylactic acid (PLA), polypropylene oxide, polyoxyethylene-polyoxypropylene copolymers, polyvinylalcohol, polyethylene glycol or polyurethanes. (1)

comprises collagen and human growth hormone, collagen and human insulin, polyethylene oxide and human growth hormone, polyethylene oxide and human insulin, carrageenan and human growth hormone or carrageenan and human insulin.

L87 ANSWER 20 OF 28 WPIX COPYRIGHT 2007 THE THOMSON CORP on STN
ACCESSION NUMBER: 2004-420518 [39] WPIX
CROSS REFERENCE: 2005-809984
DOC. NO. CPI: C2004-157940 [39]
TITLE: Composition useful for treating cancer, viral infection, bacterial infection, parasitic infection, inflammatory conditions, comprises construct having complement receptor 2 and modulator of complement activity
DERWENT CLASS: B04; D16
INVENTOR: HOLERS M V; TOMLINSON S
PATENT ASSIGNEE: (MUSC-N) MUSC FOUND RES DEV; (COLS-C) UNIV COLORADO
COUNTRY COUNT: 106

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG	MAIN IPC
WO 2004045520	A2	20040603	(200439)*	EN	184[31]	
AU 2003298650	A1	20040615	(200470)	EN		
EP 1569685	A2	20050907	(200559)	EN		
JP 2006512325	W	20060413	(200629)	JA	130	
CN 1756560	A	20060405	(200654)	ZH		A61K039-00

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2004045520	A2	WO 2003-US36459	20031113
AU 2003298650	A1	AU 2003-298650	20031113
EP 1569685	A2	EP 2003-796403	20031113
EP 1569685	A2	WO 2003-US36459	20031113
JP 2006512325	W	WO 2003-US36459	20031113
JP 2006512325	W	JP 2004-553695	20031113
CN 1756560	A	CN 2003-80108789	20031113

FILING DETAILS:

PATENT NO	KIND		PATENT NO	
AU 2003298650	A1	Based on	WO 2004045520	A
EP 1569685	A2	Based on	WO 2004045520	A
JP 2006512325	W	Based on	WO 2004045520	A

PRIORITY APPLN. INFO: US 2002-426676P 20021115
INT. PATENT CLASSIF.:
IPC ORIGINAL: A61K0038-00 [I,A]; A61K0039-395 [I,A]; A61P0001-00 [I,C]; A61P0001-04 [I,A]; A61P0011-00 [I,A]; A61P0011-06 [I,A]; A61P0013-00 [I,C]; A61P0013-12 [I,A]; A61P0015-00 [I,C]; A61P0015-08 [I,A]; A61P0017-00 [I,A]; A61P0017-02 [I,A]; A61P0019-00 [I,A]; A61P0019-02 [I,A]; A61P0021-00 [I,C]; A61P0021-04 [I,A]; A61P0025-00 [I,A]; A61P0025-28 [I,A]; A61P0027-00 [I,C]; A61P0027-02 [I,A]; A61P0029-00 [I,A]; A61P0003-00 [I,C]; A61P0003-10 [I,A]; A61P0031-00 [I,C]; A61P0031-04 [I,A]; A61P0031-06 [I,A]; A61P0031-10 [I,A]; A61P0031-12 [I,A]; A61P0031-16 [I,A]; A61P0033-00 [I,A];

A61P0033-02 [I,A]; A61P0033-04 [I,A]; A61P0033-06 [I,A]; A61P0033-12 [I,A]; A61P0035-00 [I,A]; A61P0035-02 [I,A]; A61P0037-00 [I,A]; A61P0037-02 [I,A]; A61P0043-00 [I,A]; A61P0005-00 [I,C]; A61P0005-14 [I,A]; A61P0007-00 [I,A]; A61P0007-06 [I,A]; A61P0007-08 [I,A]; A61P0009-00 [I,A]; A61P0009-10 [I,A]; C07K0014-435 [I,C]; C07K0014-47 [I,A]; C07K0014-705 [I,A]; C07K0019-00 [I,A]; A61K0039-00 [I,A]; A61K0039-395 [I,A]; C07K0014-00 [I,A]

IPC RECLASSIF.: A61K0038-00 [N,A]; A61K0038-00 [N,C]; A61K0039-00 [I,A]; A61K0039-00 [I,C]; A61K0039-395 [I,A]; A61K0039-395 [I,C] ; C07K0014-00 [I,A]; C07K0014-00 [I,C]; C07K0014-435 [I,C]; C07K0014-705 [I,A]

BASIC ABSTRACT:

WO 2004045520 A2 UPAB: 20060203

NOVELTY - A composition (C1) comprising a construct, where the construct comprises complement receptor 2 (CR2) and a modulator of complement activity.

ACTIVITY - Cytostatic; Antiasthmatic; Antiinflammatory; Dermatological; Immunosuppressive; Antiarthritic; Antirheumatic; Vasotropic; Antidiabetic; Neuroprotective; Antiallergic; Gastrointestinal-Gen; Antiulcer; Antiviral; Antibacterial; Antiparasitic (claimed).

MECHANISM OF ACTION - Inhibitor or activator of complement activity (claimed). No biological data is given.

USE - (C1) is useful for treating a condition affected by complement in a subject which involves administering (C1) to the subject. The conditions is cancer chosen from lymphomas (Hodgkins and non-Hodgkins), B cell lymphoma, T cell lymphoma, myeloid leukemia, leukemias, mycosis fungoides, carcinomas, carcinomas of solid tissues, squamous cell carcinomas, **adenocarcinomas**, sarcomas, gliomas, blastomas, neuroblastomas, plasmacytomas, histiocytomas, melanomas, adenomas, hypoxic tumors, myelomas, AIDS-related lymphomas or sarcomas, metastatic cancers, bladder cancer, brain cancer, nervous system cancer, squamous cell carcinoma of head and neck, neuroblastoma/glioblastoma, ovarian cancer, skin cancer, liver cancer, melanoma, squamous cell carcinomas of the mouth, throat, larynx, and lung, colon cancer, cervical cancer, cervical carcinoma, breast cancer, epithelial cancer, renal cancer, genitourinary cancer, pulmonary cancer, esophageal carcinoma, head and neck carcinoma, hematopoietic cancers testicular cancer, colo-rectal cancers, prostatic cancer, or pancreatic cancer. The condition is a viral infection chosen from herpes simplex virus type-1, herpes simplex virus type2, cytomegalovirus, Epstein-Barr virus, Varicella-zoster virus, Human herpesvirus 6, human herpesvirus 7, human herpesvirus 8, variola virus, vesicular stomatitis virus, hepatitis a virus, hepatitis B virus, hepatitis c virus, hepatitis D virus, hepatitis E virus, rhinovirus, coronavirus, influenza virus A, etc. The condition is bacterial infection chosen from Mycobacterium tuberculosis, M.bovis, M.bovis strain BCG, BCG substrains, M.avium, M.intracellulare, M.africanum, M.kansasii, M.marinum, M.ulcerans, M.avium subspecies paratuberculosis, Nocardia asteroides, other Nocardia species, Legionella pneumophila, other Legionella species, Salmonella typhi, other Salmonella species, Shigella species, Yersinia pestis, Pasteurella haemolytica, P.multocida, other Pasteurella species, Actinobacillus pleuropneumoniae, Listeria monocytogenes, Listeria ivanovii, Brucella abortus, other Brucella species, Cowdria ruminantium, Chlamydia pneumoniae, C.trachomatis, C.psittaci, Coxiella burnetti, other Rickettsial species, etc. The condition is parasitic infection chosen from Toxoplasma gondii, Plasmodium falciparum, P.vivax, P.malariae, other Plasmodium species, Trypanosoma brucei, T.cruzi, Leishmania major, other Leishmania species, Schistosoma mansoni, other Schistosoma species, and Entamoeba histolytica. The condition is fungal infection chosen from Candida albicans, cryptococcus neoformans, Histoplama capsulatum, Aspergillus fumigatus, Coccidiodes immitis, Paracoccidiodes brasiliensis, Blastomyces dermitidis, Pneomocystis carnii, Penicillium marneffi and Alternaria alternata. The condition is inflammatory condition

chosen from asthma, systemic lupus erythematosus, nephritis, rheumatoid arthritis, reactive arthritis, spndyarthritis, systemic vasculitis, insulin-dependent diabetes mellitus, multiple sclerosis, experimental allergic encephalomyelitis, Sjogren's syndrome, graft versus host disease, inflammatory bowel disease including Crohn's disease, ulcerative colitis, and scleroderma. The subject is a mammal which is a human or mouse. (C1) comprising fusion protein that inhibits complement is useful for reducing complement mediated damage. (C1) comprising fusion protein that activates complement is useful for enhancing complement mediated damage. (All claimed.)

DESCRIPTION OF DRAWINGS - The drawing shows inhibition of complement mediated lysis by recombinant sCD59 and CD59 fusion proteins. MANUAL CODE: CPI: B04-N08; B14-A01; B14-A02; B14-A04; B14-B02; B14-C06; B14-C09B; B14-E08; B14-E10C; B14-F02; B14-G02A; B14-G02C; B14-H01; B14-H01A; B14-H01B; B14-K01A; B14-N10; B14-N16; B14-N17; B14-S01; B14-S04; D05-C12; D05-H17C

TECH

BIOTECHNOLOGY - Preferred Composition: In (C1), the construct is a fusion protein. The fusion protein inhibits complement. The modulator of complement activity comprises a complement inhibitor which is a **decay accelerating factor** (DAF) which comprises 518 or 495 amino acids sequence fully defined in the specification. The complement inhibitor is human CD59 which comprises 330 or 334 amino acid sequence fully defined in the specification. The complement inhibitor is CR1 which has 2048 amino acid sequence fully defined in the specification. The complement inhibitor is membrane cofactor protein (MCP) which has 378 amino acid sequence fully defined in the specification. The complement inhibitor is Crry which has 440 amino acid sequence fully defined in the specification. The complement inhibitor is murine CD59. In (C1), the fusion protein activates complement. The modulator of complement activity comprises complement activator which is human immunoglobulin (Ig)G1 which has a 232 amino acid sequence fully defined in the specification. (C1) comprises 510 amino acid sequence fully defined in the specification. The complement activator is human IgM which has 454 amino acid sequence fully defined in the specification. The complement activator is mouse IgG 3 which has 233 amino acid sequence fully defined in the specification. The complement activator is CVF which has 1620 amino acid sequence fully defined in the specification. The construct of (C1) is an immunoconjugate.

L87 ANSWER 21 OF 28 EMBASE

ACCESSION NUMBER: 2006523137 EMBASE Full-text
TITLE: Bap31 enhances the endoplasmic reticulum export and quality control of human class I MHC molecules.
AUTHOR: Ladasky J.J.; Boyle S.; Seth M.; Li H.; Pentcheva T.; Abe F.; Steinberg S.J.; Edidin M.
CORPORATE SOURCE: Dr. M. Edidin, Department of Biology, Johns Hopkins University, 3400 North Charles Street, Baltimore, MD 21218, United States. edidin@jhu.edu
SOURCE: Journal of Immunology, (1 Nov 2006) Vol. 177, No. 9, pp. 6172-6181. .
Refs: 57
ISSN: 0022-1767 CODEN: JOIMA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 026 Immunology, Serology and Transplantation
029 Clinical Biochemistry

LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 22 Nov 2006
Last Updated on STN: 22 Nov 2006

ABSTRACT: The assembly of class I MHC molecules and their export from the endoplasmic reticulum (ER) is governed by chaperones and accessory proteins. We present evidence that the putative cargo receptor protein Bap31 participates in the transport and the quality control of human class I molecules. Transfection of the human adenocarcinoma cell line HeLa with yellow fluorescent protein-Bap31 chimeras increased surface levels of class I in a dose-dependent manner, by as much as 3.7-fold. The increase in surface class I resulted from an increase in the rate of export of newly synthesized class I molecules to the cell surface and from an increase in the stability of the exported molecules. We propose that Bap31 performs quality control on class I molecules in two distinct phases: first, by exporting peptide-loaded class I molecules to the ER/Golgi intermediate compartment, and second, by retrieving class I molecules that have lost peptides in the acidic post-ER environment. This function of Bap31 is conditional or redondant, because we find that Bap31 deficiency does not reduce surface class I levels. Overexpression of the Bap31 homolog, Bap29, decreases surface class levels in HeLa, indicating that it does not substitute for Bap31.

CONTROLLED TERM: Medical Descriptors:
*major histocompatibility complex
*endoplasmic reticulum
protein analysis
cell transport
quality control
protein assembly
adenocarcinoma: ET, etiology
dose response
chimera
cell surface
Golgi complex
gene overexpression
human
controlled study
human cell
article
priority journal

CONTROLLED TERM: Drug Descriptors:
*major histocompatibility antigen class 1: EC, endogenous compound
*protein BAP31: EC, endogenous compound
chaperone: EC, endogenous compound
yellow fluorescent protein
protein Bap29: EC, endogenous compound
CD9 antigen: EC, endogenous compound
decay accelerating factor: EC, endogenous compound
CD59 antigen: EC, endogenous compound
CD71 antigen: EC, endogenous compound
Fas antigen: EC, endogenous compound
CD147 antigen: EC, endogenous compound
unclassified drug

CAS REGISTRY NO.: (decay accelerating factor) 99085-47-9

L87 ANSWER 22 OF 28 EMBASE
ACCESSION NUMBER: 97072294 EMBASE Full-text

DOCUMENT NUMBER: I997072294
TITLE: Tumour necrosis factor-α up-regulates decay-acceleratiag factor gene expression in human intestinal epithelial cells.
AUTHOR: Andoh A.; Fujiyama Y.; Sumiyoshi K.; Sakumoto H.; Okabe H.; Bamba T.
CORPORATE SOURCE: Dr. A. Andoh, Department of Internal Medicine, Shiga University of Medical Science, Seta-Tukinowa, Otsu 520-21, Japan
SOURCE: Immunology, (1997) Vol. 90, No. 3, pp. 358-363. .
Refs: 35
ISSN: 0019-2805 CODEN: IMMUAM
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 022 Human Genetics
026 Immunology, Serology and Transplantation
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 4 Apr 1997
Last Updated on STN: 4 Apr 1997

ABSTRACT: The increased expression of decay-accelerating factor (DAF) has been detected in intestinal epithelial cells at the inflamed mucosa. In this study, we examined the effects of tumour necrosis factor (TNF)-α on DAF expression in three intestinal epithelial cell lines. DAF mRNA expression was evaluated by Northern blot analysis, and DAF protein expression was analysed by biotin labelling and immunoprecipitation. TNF-α induced a marked increase in DAF mRNA and protein expression in HT-29, T84 and Caco-2 cells. In HT-29 cells, the effects of TNF-α on DAF mRNA accumulation were observed in a dose-dependent manner; DAF mRNA accumulation reached a maximum at 3-6 hr, and then gradually decreased. These effects of TNF-α required de novo protein synthesis. Messenger RNA stability studies suggested that TNF-α partially regulated DAF gene expression by a posttranscriptional mechanism. Moreover, the combination of TNF-α and interleukin (IL)-4 induced an additive increase in DAF mRNA accumulation in HT-29 and T84 cells. In human intestinal epithelial cells, TNF-α acts as a potent inducer of DAF mRNA expression, indicating an important role for TNF-α in the regulation of DAF expression at the inflamed mucosa.

CONTROLLED TERM: Medical Descriptors:
*gene expression
*intestine epithelium
adenocarcinoma
article
cell line
human
human cell
priority journal
Drug Descriptors:
***decay accelerating factor: EC, endogenous compound**
*interleukin 4
*messenger rna: EC, endogenous compound
*tumor necrosis factor alpha
CAS REGISTRY NO.: (decay accelerating factor) 99085-47-9

L87 ANSWER 23 OF 28 JICST-EPlus COPYRIGHT 2007 JST on STN
ACCESSION NUMBER: 980280374 JICST-EPlus Full-text
TITLE: Expression of Membrane Cofactor Protein (CD46), **Decay Accelerating Factor (**

CD55) and Homologous Restriction Factor 20 (CD59) in Human Gastric Mucosa and Their Changes in Cancerous Tissue.

AUTHOR: SETO N
CORPORATE SOURCE: Kyoto Prefectural Univ. Medicine
SOURCE: Kyoto Furitsu Ika Daigaku Zasshi (Journal of Kyoto Prefectural University of Medicine), (1998) vol. 107, no. 2, pp. 195-206. Journal Code: Z0618A (Fig. 6, Tbl. 2, Ref. 18)
ISSN: 0023-6012
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: English
STATUS: New
ABSTRACT:

Membrane cofactor protein (MCP, CD46), **decay accelerating** ***factor*** (DAF, **CD55**) and homologous restriction factor of 20kDa (HRF20, CD59) are members of the complement regulatory proteins bound on the cell membranes which prevent the destruction of autologous cells by the attack of the complement system. These proteins exsist in some organs and on some peripheral blood cells, however the phenotypes of these proteins are different among organs or cells in the individual. The aims of this study were to determine the expression of these proteins in the gastric mucosa and the phenotypic changes in cancerous tissue. Immunohistochemical studies revealed that MCP was stained on the basolateral membrane in epithelial cells. There was no difference in the localization of MCP between normal and cancerous epithelium. MCP on cancerous epithelium was stained in like manner to normal epithelium in 6 lesions (46%) and increased in 7 (54%) out of 13 cancerous lesions. DAF wasn't stained on normal gastric epithelium but stained more intensely on cancerous epithelial cells in 7 (54%) out of 13 lesions, especially in the poorly differentiated **adenocarcinoma.** HRF20 was stained on the basal membrane from the glandular body to the base and on the lateral and apical membrane from the surface to the glandular neck. HRF20 on cancerous epithelium was stained similarly to normal epithelium in 7 (54%), diminished in 2 (15%) and increased in 4 (31%) out of 13 lesions. Western blot analysis revealed MCP was expressed with some variations in **molecular** ***weight*** (**MW**) in gastric cancer. Thus the upregulation of DAF in the cancer cells may defend against autologous complement attack more effectively. Changes of MCP's **MW** may lead to the changes of inhibitory activity against the complement and also to the appearence of neo antigen, which may be usefull in the targeting therapy as well as diagnosis. (author abst.)

CLASSIFICATION: GH04000D; GE02020C (616.3-006; 616-006.2)
CONTROLLED TERM: membrane protein; gastric mucosa; gene expression; carcinogenesis; stomach tumor; pathological state; complement(immunology); human(primates); bioactive factor
BROADER TERM: protein; stomach; gastrointestinal duct; digestive organ; mucosa; epithelial tissue; animal tissue; biomedical tissue; organization; histomembrane; membrane and film; molecular genetic phenomenon; genetic phenomenon; phenomenon; tumor process; process; stomach disease; gastrointestinal disease; digestive system disease; disease; digestive system tumor; tumor; factor

L87 ANSWER 24 OF 28 PASCAL COPYRIGHT 2007 INIST-CNRS. ALL RIGHTS RESERVED. on STN DUPLICATE 5
ACCESSION NUMBER: 1997-0144601 PASCAL Full-text

TITLE (IN ENGLISH): Complement-regulatory proteins in ovarian malignancies
AUTHOR: BJORGE L.; HAKULINEN J., WAHLSTROM T.; MATTE R.; MERI S.
CORPORATE SOURCE: Department of Bacteriology and Immunology, Haartman Institute, University of Helsinki, Helsinki, Finland; Department of Microbiology and Immunology, The Gade Institute, University of Bergen, Bergen, Norway; Department of Obstetrics and Gynecology, University Central Hospital, Helsinki, Finland
SOURCE: International journal of cancer, (1997), 70(1), 14-25, 21 refs.
ISSN: 0020-7136 CODEN: IJCNAW
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-13027, 354000062296120030
ABSTRACT: Ovarian cancer has features that makes it well-suited for MAb adjuvant immunotherapy. Several of the MAbs used in clinical trials mediate cancer cell destruction by activation of complement (C). In this study, therefore, we examined the ability of ovarian-tumor cells to resist C attack. We found that the C regulators membrane cofactor protein (MCP, CD46) and protectin (CD59) were strongly expressed in the tumor cells in all 28 benign and malignant tumors examined. **Decay-accelerating factor** (DAF; **CD55**) was more heterogeneously expressed, and only 75% of the tumors exhibited a moderate amount of DAF in the tumor cells. In adenoma cells, CD59 and DAF were preferentially located apically, while in **adenocarcinoma** cells they were expressed also at the basolateral cell surface. The ovarian-carcinoma cell lines SK-OV-3, Caov-3, SW626 and PA-1 expressed both the 58-and the 68-**kDa** isoforms of MCP. DAF was present as a glycosyl-phosphatidylinositol(GP 1)-anchored 70- **kDa glycoprotein.** The surface-expression level of DAF varied, and correlated with the vulnerability of the cells to C-mediated lysis. CD59 was expressed as a GP1-linked 19- to 25- **kDa** protein exhibiting multiple glycosylation variants. The surface expression of CD59 correlated with the amount of the main 1.9 + 2.1-kb CD59 mRNA transcripts. Neutralization of CD59 with an anti-CD59 MAb significantly enhanced C-mediated killing of the cell lines. Low expression of C regulators on the PA-1 teratocarcinoma cell line was associated with high sensitivity to C lysis. Thus, the expression of C regulators on malignant ovarian cells may constitute a tumor escape mechanism, and is a critical parameter to be examined when MAb therapy is being considered. CLASSIFICATION CODE: 002B20C02; Life sciences; Medical sciences;
Reproduction; Gynecology, Genital system; Oncology
CONTROLLED TERM: **Adenocarcinoma**; Malignant teratoma; Ovary; Complement; Membrane protein; Regulation(control); Phenotype; Gene expression; Exploration; Cytotoxicity; Human; In vivo; In vitro; Established cell line; Membrane cofactor protein; **Decay accelerating factor**
BROADER TERM: Malignant tumor; Female genital diseases; Ovarian diseases

L87 ANSWER 25 OF 28 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:521646 BIOSIS Full-text
DOCUMENT NUMBER: PREV200300510585
TITLE: Antiadhesive role of apical **decay-accelerating factor (CD55)** in human neutrophil transmigration across mucosal epithelia.
AUTHOR(S): Lawrence, Donald W.; Bruyninckx, Walter J.; Louis, Nancy A.; Lublin, Douglas M.; Stahl, Gregory L.; Parkos, Charles A.; Colgan, Sean P. [Reprint Author]

CORPORATE SOURCE: Center for Experimental Therapeutics and Reperfusion Injury, Brigham and Women's Hospital, Harvard Medical School, 20 Shattuck St., Boston, MA, 02115, USA
colgan@zeus.bwh.harvard.edu

SOURCE: Journal of Experimental Medicine, (October 6 2003) Vol. 198, No. 7, pp. 999-1010. print.
ISSN: 0022-1007 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 5 Nov 2003
Last Updated on STN: 5 Nov 2003

ABSTRACT:Neutrophil migration across mucosal epithelium during inflammatory episodes involves the precise orchestration of a number a cell surface molecules and signaling pathways. After successful migration to the apical epithelial surface, apically localized epithelial proteins may serve to retain PMN at the lumenal surface. At present, identification of apical epithelial ligands and their PMN counter-receptors remain elusive. Therefore, to define the existence of apical epithelial cell surface proteins involved in PMN-epithelial interactions, we screened a panel of antibodies directed against epithelial plasma membranes. This strategy identified one antibody (OE-1) that both localized to the apical cell membrane and significantly inhibited PMN transmigration across epithelial monolayers. Microsequence analysis revealed that OE-1 recognized human **decay-accelerating** ***factor*** (DAF, **CD55**). DAF is a highly glycosylated, 70-80-***kD*** , glycosylphosphatidyinositol-linked protein that functions predominantly as an inhibitor of autologous complement lysis. DAF suppression experiments using antisense oligonucleotides or RNA interference revealed that DAF may function as an antiadhesive molecule promoting the release of PMN from the lumenal surface after transmigration. Similarly, peptides corresponding to the antigen recognition domain of OE-1 resulted in accumulation of PMN on the apical epithelial surface. The elucidation of DAF as an apical epithelial ligand for PMN provides a target for novel anti-inflammatory therapies directed at quelling unwanted inflammatory episodes.

CONCEPT CODE: Cytology - Animal 02506
Cytology - Human 02508
Biochemistry studies - Nucleic acids, purines and pyrimidines 10062
Biophysics - Membrane phenomena 10508
Blood - Blood and lymph studies 15002
Blood - Blood cell studies 15004
Immunology - General and methods 34502
INDEX TERMS: Major Concepts
Immune System (Chemical Coordination and Homeostasis); Membranes (Cell Biology)
INDEX TERMS: Parts, Structures, & Systems of Organisms
epithelial plasma membranes; mucosal epithelium; neutrophil: blood and lymphatics, immune system, transmigration
INDEX TERMS: Chemicals & Biochemicals
OE-1 antibody; antisense oligonucleotides; apical **decay-accelerating factor** [**CD55**, DAF]: antiadhesive role; glycosylphosphatidylinositol-linked protein
INDEX TERMS: Methods & Equipment
RNA interference: genetic techniques, laboratory techniques; microsequence analysis: genetic techniques, laboratory techniques
ORGANISM: Classifier
Cricetidae 86310
Super Taxa

Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
CHO cell line (cell line): Chinese hamster ovary cells
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

ORGANISM:
Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
CaCo2 cell line (cell line): human colona **adenocarcinoma** cells
HMVEC cell line (cell line): human microvascular endothelial cells
KB cell line (cell line): human squamous cell mouth carcinoma cells
OKF6 cell line (cell line): human oral mucosal epithelial cells
T84 cell line (cell line): human colon **adenocarcinoma** cells
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates

L87 ANSWER 26 OF 28 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2005:882482 SCISEARCH Full-text
THE GENUINE ARTICLE: 954HZ
TITLE: Helicobacter pylori eradication decreases the expression of glycosylphosphatidylinositol-anchored complement regulators, **decay-accelerating factor** and homologous restriction factor 20, in human gastric epithelium
AUTHOR: Joh T; Sasaki M (Reprint); Kataoka H; Tanida S; Itoh K; Kondo Y; Ogasawara N; Oshima T; Okada N; Ohara H; Sano H; Nakao H; Sobue S; Itoh M
CORPORATE SOURCE: Nagoya City Univ, Grad Sch Med Sci, Dept Internal Med & Bioregulat, Mizuho Ku, 1 Kawasumi, Nagoya, Aichi 467, Japan (Reprint); Nagoya City Univ, Grad Sch Med Sci, Dept Internal Med & Bioregulat, Mizuho Ku, Nagoya, Aichi 467, Japan; Nagoya City Univ, Grad Sch Med Sci, Dept Mol Biol, Nagoya, Aichi, Japan
msasaki@med.nagoya-cu.ac.jp
COUNTRY OF AUTHOR: Japan
SOURCE: JOURNAL OF GASTROENTEROLOGY AND HEPATOLOGY, (SEP 2005) Vol. 20, No. 9, pp. 1344-1351.
ISSN: 0815-9319.
PUBLISHER: BLACKWELL PUBLISHING, 9600 GARSINGTON RD, OXFORD OX4 2DQ, OXON, ENGLAND.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 37
ENTRY DATE: Entered STN: 8 Sep 2005
Last Updated on STN: 8 Sep 2005

ABSTRACT:
Background: It has previously been reported that there is a strong correlation between the expression of glycosylphosphatidylinositol (GPI)-anchored complement membrane inhibitor in gastric epithelium and the severity of inflammation of gastric mucosa. To investigate the regulation of

complement activity in gastric epithelium during Helicobacter pylori (H. pylori)-associated gastritis, the expression of GPI-anchored complement membrane inhibitors, **decay-accelerating factor** (DAF) and 20-**kDa** homologous restriction factor 20 (HRF20), and membrane cofactor protein (MCP), which is a transmembrane protein, were evaluated after removal of the H. pylori stimulus. Furthermore, the expression of the complement fragment, C3c, was also investigated.

Methods: Forty-six patients with epigastric symptoms and endoscopically confirmed peptic ulcer or gastritis who had H. pylori infection of the gastric mucosa were enrolled in the present study. Biopsy specimens were obtained from the gastric antrum and corpus 1 month before and after eradication. Helicobacter pylori infection was determined by the rapid urease test, histology, and culture before eradication, and by histology, culture, and urea breath test after eradication. Gastric biopsy specimens obtained before and after eradication were evaluated for infiltration by neutrophils and mononuclear cells. The expression of complement membrane inhibitors, DAF, HRF20, and MCP and that of the main complement fragment, C3c, was immunohistochemically evaluated.

Results: One month after the eradication of H. pylori, the infiltration by neutrophils and mononuclear cells in the gastric mucosa decreased significantly ($P < 0.0001$) as compared with that before eradication. The expression of DAF, HRF20, and C3c on gastric mucosal epithelium also significantly decreased in both the antrum and the corpus ($P < 0.05$) 1 month after eradication. However, no change was observed in the expression of MCP.

Conclusions: The decrease in the expression of GPI-anchored complement regulator and the complement after removal of a chronic microbial stimulus suggests that the gastric epithelium appears to undergo an aggressive stress of complement during H. pylori infection. Conclusively, DAF and HRF20 may play an important protective role against complement-mediated damage induced by chronic microbial stimuli in such a pathological condition.

CATEGORY: GASTROENTEROLOGY & HEPATOLOGY

SUPPLEMENTARY TERM: C3c; complement activation; DAF; eradication; Helicobacter pylori; HRF20

SUPPL. TERM PLUS: MEMBRANE COFACTOR PROTEIN; **ADENOCARCINOMA** CELL-LINE; ENDOTHELIAL-CELLS; FACTOR DAF; C-3 CONVERTASES; CD59; PHOSPHOLIPASE; C5B-9; MICE

REFERENCE(S):

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	ARN PG (RPG)	Referenced Work (RWK)
BALDWIN W M	2003	25	181	SPRINGER SEMIN IMMUN
BERSTAD A E	1998	42	522	GUT
BERSTAD A E	1997	40	196	GUT
BERSTAD A E	2001	120	1108	GASTROENTEROLOGY
BHAKDI S	1988	74	459	CLIN EXP IMMUNOL
BJORGE L	1996	42	185	CANCER IMMUNOL IMMUN
BJORGE L	1995	41	350	SCAND J IMMUNOL
BORN J	1986	59	139	IMMUNOLOGY
BROWN E J	1983	6	349	SPRINGER SEMIN IMMUN
CHMIELA M	1997	40	20	GUT
DAVITZ M A	1987	715	111	ACTA MED SCAND S
DAVITZ M A	1986	163	1150	J EXP MED
DORRELL N	1999	117	1098	GASTROENTEROLOGY
FUJITA T	1987	166	1221	J EXP MED
INOUE T	2002	55	193	J CLIN PATHOL-MOL PA
ISMAIL H F	2003	71	7140	INFECT IMMUN
KAWANO M	2000	48	367	ARCH IMMUNOL THER EX
KINOSHITA T	1991	12	291	IMMUNOL TODAY
KOOYMAN D L	1995	269	89	SCIENCE

MCNEARNEY T	1989	84	538	J CLIN INVEST
MEDOF M E	1987	165	848	J EXP MED
MEDOF M E	1986	25	6740	BIOCHEMISTRY-US
MERI S	1993	23	2511	EUR J IMMUNOL
MIWA T	2001	1	445	INT IMMUNOPHARMACOL
MOUTABARRIK A	1993	12	167	LYMPHOKINE CYTOK RES
NICHOLSONWELLER A	1982	129	184	J IMMUNOL
NOSE M	1990	70	145	IMMUNOLOGY
OKADA N	1989	1	205	INT IMMUNOL
OSHIMA T	2000	164	1078	J IMMUNOL
RAUTEMAA R	2001	120	470	GASTROENTEROLOGY
ROKITA E	1998	178	1521	J INFECT DIS
ROLLINS S A	1990	144	3478	J IMMUNOL
SASAKI M	1998	33	554	HISTOPATHOLOGY
SEYA T	1986	163	837	J EXP MED
SJUNNESSON H	2001	30	167	FEMS IMMUNOL MED MIC
SOHN J H	2000	41	4195	INVEST OPHTH VIS SCI
VAKEVA A	1994	82	28	IMMUNOLOGY

L87 ANSWER 27 OF 28 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1997:813137 SCISEARCH Full-text

THE GENUINE ARTICLE: YD878

TITLE: Expression of the complement regulatory proteins **decay accelerating factor** (DAF, **CD55**), membrane cofactor protein (MCP, CD46) and CD59 in the normal human uterine cervix and in premalignant and malignant cervical disease

AUTHOR: Simpson K L (Reprint); Jones A; Norman S; Holmes C H

CORPORATE SOURCE: UNIV BRISTOL, ST MICHAELS HOSP, DEPT CLIN MED, DIV OBSTET & GYNAECOL, BRISTOL BS2 8EG, AVON, ENGLAND

COUNTRY OF AUTHOR: ENGLAND

SOURCE: AMERICAN JOURNAL OF PATHOLOGY, (NOV 1997) Vol. 151, No. 5, pp. 1455-1467.
ISSN: 0002-9440.

PUBLISHER: AMER SOC INVESTIGATIVE PATHOLOGY, INC, 428 EAST PRESTON ST, BALTIMORE, MD 21202-3993.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE; CLIN

LANGUAGE: English

REFERENCE COUNT: 51

ENTRY DATE: Entered STN: 1997
Last Updated on STN: 1997

ABSTRACT:

The membrane-bound complement regulators **decay-** ***accelerating*** **factor** (DAF, **CD55**), membrane cofactor protein (MCP, CD46), and CD59 are broadly expressed proteins that act together to protect host tissues from autologous complement. Comparison of expression profiles of these proteins between normal and pathological tissues could reveal a mechanism by which tumor cells evade complement-mediated killing, Expression of the regulators was therefore examined in the normal human uterine cervix, in cervical intraepithelial neoplasia (CIN; n = 23), and in cervical squamous carcinomas (n = 6), DAF and MCP were reciprocally expressed in normal ectocervical epithelium. MCP was confined predominantly to the basal and parabasal layers with more extensive expression in metaplastic squamous epithelium. An apparent expansion in MCP expression was observed in more severe premalignant lesions whereas cervical carcinomas were uniformly MCP positive. By contrast, DAF expression appeared unaltered in premalignant lesions and variable in carcinomas. However, increased DAF was observed in stromal cells directly adjacent to infiltrating tumor cells. A low

molecular **weight** DAF-product was detected in tumors, and preliminary evidence suggests this may be derived from stromal cells, Overall, changes in expression of C3 convertase regulators in both the stromal and epithelial compartments may be important for evasion of immune surveillance in cervical cancer.

CATEGORY: PATHOLOGY

SUPPL. TERM PLUS: SIGNAL-TRANSDUCING MOLECULE; **ADENOCARCINOMA** CELL-LINE; ENDOTHELIAL-CELLS; FETOMATERNAL INTERFACE; HOMOLOGOUS COMPLEMENT; MEASLES-VIRUS; HUMAN TISSUES; RECEPTOR; SYSTEM; IDENTIFICATION

REFERENCE(S):

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	ARN PG (RPG)	Referenced Work (RWK)
ATKINSON J P	1987	8	212	IMMUNOL TODAY
BERGELSON J M	1994	91	6245	P NATL ACAD SCI USA
BJORGE L	1996	42	185	CANCER IMMUNOL IMMUN
BJORGE L	1997	70	14	INT J CANCER
BJORGE L	1995	41	350	SCAND J IMMUNOL
BORA N S	1991	146	2821	J IMMUNOL
BRYANT R W	1991	147	1856	J IMMUNOL
CERVONI F	1993	151	939	J IMMUNOL
CHEUNG N K V	1988	81	1122	J CLIN INVEST
CINEK T	1992	149	2262	J IMMUNOL
CUI W	1994	8	A765	FASEB J
DAVIS L S	1988	141	2246	J IMMUNOL
DORIG R E	1993	75	295	CELL
FLETCHER A	1992	75	507	IMMUNOLOGY
HOLMES C H	1992	22	1579	EUR J IMMUNOL
HOLMES C H	1990	144	3099	J IMMUNOL
JENSEN T S	1995	34	1	AM J REPROD IMMUNOL
KINOSHITA T	1991	12	291	IMMUNOL TODAY
KORTY P E	1991	146	4092	J IMMUNOL
KUMAR S	1993	53	348	CANCER RES
LAEMMLI U K	1970	227	680	NATURE
LISZEWSKI M K	1994	269	10776	J BIOL CHEM
LUBLIN D M	1989	7	35	ANNU REV IMMUNOL
MCNEARNEY T	1989	84	538	J CLIN INVEST
MEDOF M E	1987	165	848	J EXP MED
MERI S	1990	71	1	IMMUNOLOGY
MERI S	1991	65	532	LAB INVEST
NANICHE D	1993	67	6025	J VIROL
NIEHANS G A	1996	149	129	AM J PATHOL
NOSE M	1990	70	145	IMMUNOLOGY
NOWICKI B	1993	178	2115	J EXP MED
OGLESBY T J	1996	246	78	ANAT REC
OKADA N	1995	92	2489	P NATL ACAD SCI USA
PESANDO J M	1987	19	235	HUM IMMUNOL
PRICE R J	1979	32	61	FERTIL STERIL
ROLLINS S A	1991	146	2345	J IMMUNOL
RUSSELL S M	1992	22	1513	EUR J IMMUNOL
SAYAMA K	1992	127	1	BRIT J DERMATOL
SAYAMA K	1991	96	61	J INVEST DERMATOL
SEYA T	1989	264	581	BIOCHEM J
SEYA T	1990	145	238	J IMMUNOL
SHIBATA T	1991	147	3901	J IMMUNOL
SHINOURA N	1994	86	143	CANCER LETT
SIMPSON K L	1993	80	183	IMMUNOLOGY
SIMPSON K L	1994	81	452	IMMUNOLOGY
SMITH N C	1974	252	302	NATURE

SPARROW R L	1983	7	1	HUM IMMUNOL
SUNDERLAND C A	1984	44	4406	CANCER RES
TOWBIN H	1979	76	4350	P NATL ACAD SCI USA
TSUJI S	1993	152	1404	J IMMUNOL
YAMAKAWA M	1994	73	2808	CANCER

L87 ANSWER 28 OF 28 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 1994:452006 SCISEARCH Full-text
THE GENUINE ARTICLE: NX261
TITLE: EXPRESSION AND FUNCTION OF CD59 ON COLONIC **ADENOCARCINOMA** CELLS
AUTHOR: BJORGE L (Reprint); VEDELER C A; ULVESTAD E; MATRE R
CORPORATE SOURCE: GADE INST, DEPT MICROBIOL & IMMUNOL, ARMAUER HANSEN BLDG, N-5021 BERGEN, NORWAY (Reprint); UNIV BERGEN, BROEGELMANN RES LAB MICROBIOL, BERGEN, NORWAY; UNIV BERGEN, DEPT NEUROL, N-5014 BERGEN, NORWAY
COUNTRY OF AUTHOR: NORWAY
SOURCE: EUROPEAN JOURNAL OF IMMUNOLOGY, (JUL 1994) Vol. 24, No. 7, pp. 1597-1603.
ISSN: 0014-2980.
PUBLISHER: VCH PUBLISHERS INC, 303 NW 12TH AVE, DEERFIELD BEACH, FL 33442-1788.
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FILE SEGMENT: LIFE
LANGUAGE: English
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ENTRY DATE: Entered STN: 1994
Last Updated on STN: 1994

ABSTRACT:
The expression and function of CD59, a 19-25 **kDa** membrane ***glycoprotein*** that inhibits formation of the membrane attack complex of complement,was analyzed on normal and malignant human colonic epithelial cells. Analysis by immuno-fluorescence demonstrated a weak apical expression of CD59 on normal intestinal epithelium, with an increased expression on ***adenocarcinoma*** cells. The expression of CD59 was greatest on tumor cells with poor differentiation. The functional activity of CD59 on human ***adenocarcinoma*** cells was investigated using the colonic ***adenocarcinoma*** cell line HT29. CD59 on HT29 cells was glycosyl-phosphatidylinositol-linked, and had a molecular mass of 19-25 ***kDa.*** HT29 cells expressed approximately four times more CD59 than leukocytes, and showed a high resistance to antibody-dependent complement-mediated lysis. Blocking of CD59 with divalent antigen-binding F(ab')(2) fragments of the anti-CD59 monoclonal antibody 1F5 resulted in a dose-dependent increase in complement-mediated lysis, suggesting that CD59 may be of importance in protecting colonic **adenocarcinoma** cells against complement-mediated cytolysis.

CATEGORY: IMMUNOLOGY
SUPPLEMENTARY TERM: COMPLEMENT; CD59; HUMAN COLORECTAL **ADENOCARCINOMAS**
SUPPL. TERM PLUS: **DECAY-ACCELERATING FACTOR**; MEMBRANE ATTACK COMPLEX; HUMAN-ERYTHROCYTE MEMBRANE; NORMAL HUMAN TISSUES; TRANSMEMBRANE CHANNELS; HOMOLOGOUS COMPLEMENT; INHIBITING PROTEIN; EPITHELIAL-CELLS; TUMOR-CELLS; COFACTOR

REFERENCE(S):

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	ARN PG (RPG)	Referenced Work (RWK)
ARULANANDAM, A R N	1993	177	1439	J EXP MED
BJORGE, L	1993	36	233	IMMUNOL LETT

BROOIMANS, E A	1992	22	791	EUR J IMMUNOL
CHEUNG, N K V	1988	81	1122	J CLIN INVEST
DAVIES, A	1989	170	637	J EXP MED
DECKERT, M	1992	22	2943	EUR J IMMUNOL
DECKERT, M	1992	148	672	J IMMUNOL
FEARON, D T	1979	76	5867	P NATL ACAD SCI USA
HOLGUIN, M H	1989	184	7	J CLIN INVEST
HOLMES, C H	1992	22	1579	EUR J IMMUNOL
IRIE, K	1974	186	454	SCIENCE
JOHNSTONE, R W	1993	79	341	IMMUNOLOGY
KUMAR, S	1993	53	348	CANCER RES
LAEMMLI, U K	1970	227	680	NATURE
LISANTI, M P	1990	15	113	TRENDS BIOCHEM SCI
LUBLIN, D M	1991	174	35	J EXP MED
MERI, S	1990	71	1	IMMUNOLOGY
MERI, S	1991	65	532	LAB INVEST
MORGAN, B P	1992	282	409	BIOCHEM J
MULLEREBERHARD, H J	1985		147	COMPLEMENT
NICHOLSONWELLER, A	1982	129	184	J IMMUNOL
NOSE, M	1990	70	145	IMMUNOLOGY
OKADA, N	1989	1	205	INT IMMUNOL
OLD, L J	1981	41	361	CANCER RES
PANNEERSELVAM, M	1986	136	2534	J IMMUNOL
PARHAM, P	1983	131	2895	J IMMUNOL
RATNOFF, W D	1992	87	415	CLIN EXP IMMUNOL
ROONEY, I A	1991	83	251	CLIN EXP IMMUNOL
ROONEY, I A	1992	76	541	IMMUNOLOGY
SCHONERMARK, S	1986	136	1772	J IMMUNOL
SEYA, T	1986	163	837	J EXP MED
SEYA, T	1990	145	238	J IMMUNOL
SIMS, P J	1989	264	19228	J BIOL CHEM
STEFANOVA, I	1989	26	153	MOL IMMUNOL
SUGITA, Y	1988	104	633	J BIOCH
TANDON, N	1992	75	372	IMMUNOLOGY
TARTAKOFF, A M	1992	17	470	TRENDS BIOCHEM SCI
TERACHI, T	1991	51	2521	CANCER RES
VLOCK, D R	1989	49	1361	CANCER RES
WALSH, L A	1992	40	213	TISSUE ANTIGENS
ZALMAN, L S	1986	83	6975	P NATL ACAD SCI USA

FILE 'HOME' ENTERED AT 17:03:23 ON 17 JAN 2007

SEARCH HISTORY

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=> d his nofile

     (FILE 'HOME' ENTERED AT 14:56:16 ON 17 JAN 2007)

     FILE 'CAPLUS' ENTERED AT 14:56:31 ON 17 JAN 2007
L1           48 SEA ABB=ON  VOLLMERS H?/AU
L2         5844 SEA ABB=ON  MUELLER HERMELINK H?/AU OR MUELLER H?/AU OR
                  HERMELINK H?/AU
L3           25 SEA ABB=ON  L1 AND L2
L4          362 SEA ABB=ON  CD55/OBI OR CD 55/OBI
L5            2 SEA ABB=ON  L3 AND L4
                  D SCAN
L6        21213 SEA ABB=ON  CARCINOMA#/OBI(L)ADENO/OBI OR ADENOCARCINOMA#/OBI
L7            4 SEA ABB=ON  23132/OBI
L8            0 SEA ABB=ON  L4 AND L7
L9            4 SEA ABB=ON  (L1 OR L2) AND L7
                  D SCAN
L10           6 SEA ABB=ON  L4 AND L6

     FILE 'REGISTRY' ENTERED AT 15:01:37 ON 17 JAN 2007
L11           1 SEA ABB=ON  99085-47-9

     FILE 'REGISTRY' ENTERED AT 15:01:58 ON 17 JAN 2007
                  D IDE

     FILE 'CAPLUS' ENTERED AT 15:02:41 ON 17 JAN 2007
L12         967 SEA ABB=ON  DECAY-ACCELERATING FACTOR/OBI
L13        1086 SEA ABB=ON  L11
L14          18 SEA ABB=ON  L6 AND (L4 OR L12 OR L13)
L15          12 SEA ABB=ON  L6 AND L12
L16          16 SEA ABB=ON  L6 AND L13
L17           8 SEA ABB=ON  L6 (L)(L12 OR L13)
L18          12 SEA ABB=ON  L6 AND L12 AND L13
                  D QUE
L19           3 SEA ABB=ON  L18 NOT (L5 OR L9 OR L10 OR L17)
L20      114772 SEA ABB=ON  GLYCOPROTEIN#/OBI
L21         177 SEA ABB=ON  PROTEIN#/OBI(L)GLYCO/OBI
                  D SCAN L19
L22           6 SEA ABB=ON  L6 AND (L20 OR L21) AND (L12 OR L13)

     FILE 'MEDLINE' ENTERED AT 15:07:57 ON 17 JAN 2007
L23         674 SEA ABB=ON  MUELLER HERMELINK H?/AU OR MUELLER H?/AU OR
                  HERMELINK H?/AU
L24          50 SEA ABB=ON  VOLLMERS H?/AU
L25           1 SEA ABB=ON  L23 AND L24
                  D TRIAL
L26        1166 SEA ABB=ON  ANTIGENS, CD55/CT
L27       53324 SEA ABB=ON  GLYCOPROTEINS/CT
L28      203676 SEA ABB=ON  ADENOCARCINOMA+NT/CT
L29          24 SEA ABB=ON  23132
L30      393194 SEA ABB=ON  CELL LINE+NT/CT
L31           3 SEA ABB=ON  L29 AND (L30 OR L28)
L32           0 SEA ABB=ON  L26 AND L29
                  D TRIAL L31 1-3
                  D KWIC L31 1-3
L33           5 SEA ABB=ON  L26 AND L28 AND L30
L34          16 SEA ABB=ON  L26 AND L28
L35           0 SEA ABB=ON  L26 AND L28 AND L27
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L36            11 SEA ABB=ON  L34 NOT (L33 OR L25)
                  D TRIAL 1-11
                  D QUE
                  D TRIAL 1-11

     FILE 'STNGUIDE' ENTERED AT 15:26:01 ON 17 JAN 2007
                  D QUE
                  D QUE L34

     FILE 'EMBASE' ENTERED AT 15:28:33 ON 17 JAN 2007
L37            53 SEA ABB=ON  VOLLMERS H?/AU
L38           896 SEA ABB=ON  MUELLER HERMELINK H?/AU OR MUELLER H?/AU OR
                  HERMELINK H?/AU
                  E CD55/CT
                  E E3+ALL
                  E E2+ALL
L39          1317 SEA ABB=ON  DECAY ACCELERATING FACTOR/CT
L40             5 SEA ABB=ON  23132?
                  E ADENOCARCINOMA/CT
                  E E3+ALL
L41         16843 SEA ABB=ON  ADENOCARCINOMA/CT
                  E CELL LINE+ALL/CT
L42         42527 SEA ABB=ON  CELL LINE/CT
L43          9850 SEA ABB=ON  TUMOR CELL LINE/CT
                  E GLYCOPROTEIN/CT
                  E E3+ALL
L44        217084 SEA ABB=ON  GLYCOPROTEIN+NT/CT
L45         27881 SEA ABB=ON  GLYCOPROTEIN/CT
L46             8 SEA ABB=ON  (L37 AND L38) OR ((L37 OR L38) AND L39)
                  D TRIAL 1-8

     FILE 'STNGUIDE' ENTERED AT 16:30:00 ON 17 JAN 2007

     FILE 'EMBASE' ENTERED AT 16:30:41 ON 17 JAN 2007
L47             3 SEA ABB=ON  L39 AND L41
L48             7 SEA ABB=ON  L39 AND (L42 OR L43) AND L44
L49             0 SEA ABB=ON  L39 AND (L42 OR L43) AND L45
                  D TRIAL L48 1-7
                  D HS

     FILE 'WPIX' ENTERED AT 16:31:44 ON 17 JAN 2007
L50            14 SEA ABB=ON  VOLLMERS H?/AU
L51          2606 SEA ABB=ON  MUELLER HERMELINK H?/AU OR MUELLER H?/AU OR
                  HERMELINK H?/AU
L52           119 SEA ABB=ON  CD55/BI,ABEX OR CD 55/BI,ABEX OR DECAY ACCELERATING
                   FACTOR/BI,ABEX
L53             1 SEA ABB=ON  (L50 AND L51) OR ((L50 OR L51) AND L52)
                  D TRIAL
                  D BIB
L54             0 SEA ABB=ON  MUELLER-HERMELINK H?/AU
                  D TRIAL L53

     FILE 'STNGUIDE' ENTERED AT 16:34:14 ON 17 JAN 2007

     FILE 'WPIX' ENTERED AT 16:35:04 ON 17 JAN 2007
                  E B04-N06+ALL/MC
                  E B11-C08E+ALL/MC
                  E B12-K04A1+ALL/MC
                  E D05-H09+ALL/MC
                  E D05-H13+ALL/MC
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     FILE 'EMBASE' ENTERED AT 16:36:07 ON 17 JAN 2007
L55           0 SEA ABB=ON  L39 AND L40

     FILE 'WPIX' ENTERED AT 16:36:21 ON 17 JAN 2007
L56        3675 SEA ABB=ON  B04-N06/MC OR C04-N06/MC
L57        6999 SEA ABB=ON  GLYCOPROTEIN#/BI,ABEX OR GLYCO PROTEIN#/BI,ABEX
L58        3007 SEA ABB=ON  ADENOCARCINOMA#/BI,ABEX OR ADENO/BI,ABEX(A)CARCINOM
                A#/BI,ABEX
L59           5 SEA ABB=ON  23132?/BI,ABEX
L60           4 SEA ABB=ON  L52 AND L58
L61           1 SEA ABB=ON  L52 AND L59
L62          20 SEA ABB=ON  L52 AND (L56 OR L57)
L63      220303 SEA ABB=ON  MW/BI,ABEX OR MOL?/BI,ABEX(W)WEIGHT/BI,ABEX OR
                KDA/BI,ABEX OR DALTON#/BI,ABEX OR KILODALTON#/BI,ABEX OR
                KD/BI,ABEX
L64           2 SEA ABB=ON  L62 AND L63
L65      132265 SEA ABB=ON  82/BI,ABEX OR 82000/BI,ABEX
L66           2 SEA ABB=ON  (L65 OR L63) AND L62

     FILE 'STNGUIDE' ENTERED AT 16:40:18 ON 17 JAN 2007

     FILE 'JICST-EPLUS, PASCAL, BIOTECHNO, BIOSIS, ESBIOBASE, BIOTECHDS,
     LIFESCI, CONFSCI, DISSABS, BIOENG, SCISEARCH' ENTERED AT 16:42:21 ON 17
     JAN 2007
L67         306 SEA ABB=ON  VOLLMERS H?/AU
L68       10926 SEA ABB=ON  MUELLER HERMELINK H?/AU OR MUELLER H?/AU OR
                HERMELINK H?/AU
L69        7655 SEA ABB=ON  CD55 OR CD 55 OR DECAY ACCELERATING FACTOR
L70      546531 SEA ABB=ON  GLYCOPROTEIN# OR GLYCO PROTEIN#
L71          51 SEA ABB=ON  23132?
L72      225456 SEA ABB=ON  ADENOCARCINOMA# OR ADENO(A) CARCINOMA#
L73     1412828 SEA ABB=ON  MW OR MOL?(W) WEIGHT OR KDA OR DALTON# OR KILODALTO
                N# OR KD OR 82 OR 82000
L74         136 SEA ABB=ON  (L67 AND L68) OR ((L67 OR L68) AND (L69 OR L71))
L75         113 SEA ABB=ON  (L67 AND L68)
L76          14 SEA ABB=ON  (L67 AND L68) AND (L69 OR L71)
L77           1 SEA ABB=ON  L69 AND L71
L78         103 SEA ABB=ON  L69 AND L72
L79          18 SEA ABB=ON  L78 AND (L70 OR L73)

     FILE 'STNGUIDE' ENTERED AT 16:50:09 ON 17 JAN 2007

     FILE 'JICST-EPLUS, PASCAL, BIOTECHNO, BIOSIS, ESBIOBASE, BIOTECHDS,
     LIFESCI, CONFSCI, DISSABS, BIOENG, SCISEARCH' ENTERED AT 16:54:35 ON 17
     JAN 2007
               D QUE L76

     FILE 'MEDLINE' ENTERED AT 16:54:37 ON 17 JAN 2007
               D QUE L25

     FILE 'EMBASE' ENTERED AT 16:54:38 ON 17 JAN 2007
               D QUE L46

     FILE 'WPIX' ENTERED AT 16:54:39 ON 17 JAN 2007
               D QUE L53

     FILE 'CAPLUS' ENTERED AT 16:54:40 ON 17 JAN 2007
               D QUE L5
               D QUE L9
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L80          6 SEA ABB=ON  L5 OR L9

     FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE' ENTERED AT 16:55:03 ON 17 JAN 2007

     FILE 'MEDLINE, CAPLUS, WPIX, PASCAL, BIOSIS, BIOTECHDS, EMBASE' ENTERED
     AT 16:58:40 ON 17 JAN 2007
L81         22 DUP REM L25 L80 L53 L76 L46 (8 DUPLICATES REMOVED)
                    ANSWER '1' FROM FILE MEDLINE
                    ANSWERS '2-7' FROM FILE CAPLUS
                    ANSWER '8' FROM FILE WPIX
                    ANSWERS '9-11' FROM FILE PASCAL
                    ANSWERS '12-15' FROM FILE BIOSIS
                    ANSWER '16' FROM FILE BIOTECHDS
                    ANSWERS '17-22' FROM FILE EMBASE
               D IBIB ED ABS 1-22

     FILE 'STNGUIDE' ENTERED AT 16:59:09 ON 17 JAN 2007

     FILE 'MEDLINE' ENTERED AT 17:01:51 ON 17 JAN 2007
               D QUE L32
               D QUE L35
               D QUE L33
L82           5 SEA ABB=ON  L33 NOT L25

     FILE 'EMBASE' ENTERED AT 17:01:52 ON 17 JAN 2007
               D QUE L47
               D QUE L49
               D QUE L55
L83           3 SEA ABB=ON  L47 NOT L46

     FILE 'WPIX' ENTERED AT 17:01:54 ON 17 JAN 2007
               D QUE L60
               D QUE L61
               D QUE L66
L84           4 SEA ABB=ON  (L60 OR L61 OR L66) NOT L53

     FILE 'CAPLUS' ENTERED AT 17:01:57 ON 17 JAN 2007
               D QUE L10
               D QUE L17
               D QUE L22
               D QUE L8
L85          14 SEA ABB=ON  (L10 OR L17 OR L22) NOT L80

     FILE 'JICST-EPLUS, PASCAL, BIOTECHNO, BIOSIS, ESBIOBASE, BIOTECHDS,
     LIFESCI, CONFSCI, DISSABS, BIOENG, SCISEARCH' ENTERED AT 17:01:59 ON 17
     JAN 2007
               D QUE L77
               D QUE L79
L86           15 SEA ABB=ON  (L77 OR L79) NOT L76

     FILE 'STNGUIDE' ENTERED AT 17:02:11 ON 17 JAN 2007

     FILE 'MEDLINE, CAPLUS, WPIX, EMBASE, JICST-EPLUS, PASCAL, BIOTECHNO,
     BIOSIS, ESBIOBASE, SCISEARCH' ENTERED AT 17:02:44 ON 17 JAN 2007
L87           28 DUP REM L82 L85 L84 L83 L86 (13 DUPLICATES REMOVED)
                    ANSWERS '1-5' FROM FILE MEDLINE
                    ANSWERS '6-18' FROM FILE CAPLUS
                    ANSWERS '19-20' FROM FILE WPIX
                    ANSWERS '21-22' FROM FILE EMBASE
                    ANSWER '23' FROM FILE JICST-EPLUS
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ANSWER '24' FROM FILE [illegible]
ANSWER '25' FROM FILE BIOSIS
ANSWERS '26-28' FROM FILE SCISEARCH
D IALL 1-5
D IBIB ED ABS HITIND 6-18
D IALL ABEQ TECH 19-20
D IALL 21-28

FILE 'HOME' ENTERED AT 17:03:23 ON 17 JAN 2007

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